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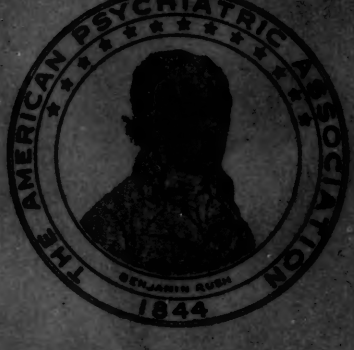

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THE AUTONOMIC NERVOUS SYSTEM.

A Sketch of Its Structure and Functions with a Consideration of Its Cerebral Connections.*

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Introduction.

The objects of this paper are to give a summarized account of the structure and functions of the autonomic system, followed by a description of some recent experimental evidence of the presence of centres in the hypothalamic area of the diencephalon, which are concerned with the control of the sympathetic and para-sympathetic divisions of this system. The paper will be illustrated by some examples of pathological states of the hypothalamus in man.

The Autonomic Nervous System.

Structure.

Three sets of ganglia containing autonomic nerve-cells are found in the animal body. The first set, the paravertebral ganglia, are found as ganglionic enlargements of the autonomic nerve-cords, which extend on each side of the vertebral column from the base of the skull to the anterior aspect of the coccyx. The prevertebral ganglia make up the second set of autonomic nerve-cells and are situated in the structures known anatomically as the cardiac, cœliac and hypogastric plexuses. Finally, collections of autonomic nerve cells are found in close proximity with, if not within, the organ which they are destined to supply. These make up the terminal set of autonomic ganglia. The axone of every autonomic nerve-cell is believed to terminate peripherally in the involuntary musculature or glandular tissue of the viscera and to carry effector nerve impulses to these structures. The axone of the autonomic nerve-cell is generally spoken of as the post-ganglionic fibre.

*Read before the Academy of Medicine, Hamilton, Ontario, November 15, 1933.

Each autonomic nerve-cell is similarly believed to be connected with the central nervous system by a neurone, the cell body of which lies in the central nervous tissue of the brain or spinal cord. This cell has been named the connector cell and its axone, which leaves the central nervous system to synapse around an autonomic nerve-cell, has been called the connector fibre. This fibre is also frequently termed the pre-ganglionic fibre.

It, therefore, follows that the nerve pathway from the central nervous system to the viscus is made up of two neurones; the connector cell with its axone, or pre-ganglionic fibre, and the autonomic nerve-cell with its axone or postganglionic fibre. (Fig. 1.)

Gaskell [1] published in 1886 the results of his investigations of the distribution of connector fibres. He showed them to be small medullated fibres which left the central nervous system in three situations: (1) in the substance of the third, seventh, ninth and tenth cranial nerves, (2) in the ventral roots of the second thoracic to the second lumbar spinal nerves and, (3) in the ventral roots of the second, third and fourth sacral nerves. The histological appearances of connector fibres can be seen in the photomicrographs of a transverse section of the ventral root of a human fifth thoracic nerve. (Figs. 2 and 3.) The distribution of the cranial and sacral outflows of connector fibres is shown in Fig. 4 and this is usually spoken of as the parasympathetic division of the autonomic nervous system. Gaskell's thoracico-lumbar outflow of connector fibres is shown in Fig. 5 and this makes up the sympathetic division of the autonomic nervous system. Gaskell also noted that the distribution of the thoracico-lumbar or sympathetic outflow overlapped that of the cranial outflow above and of the sacral outflow below. He consequently postulated that the visceral muscular and glandular tissue throughout the body received a double innervation: (1) from the sympathetic outflow and, (2) from either the cranial or the sacral portions of the parasympathetic outflow. He suggested that these two innervations were antagonistic in their action on the viscus supplied, and that impulses arriving over the cranial and sacral, or parasympathetic, outflows were mainly anabolic in their function, whereas sympathetic impulses reaching the viscus through the thoracico-lumbar outflow, exerted a katabolic effect.

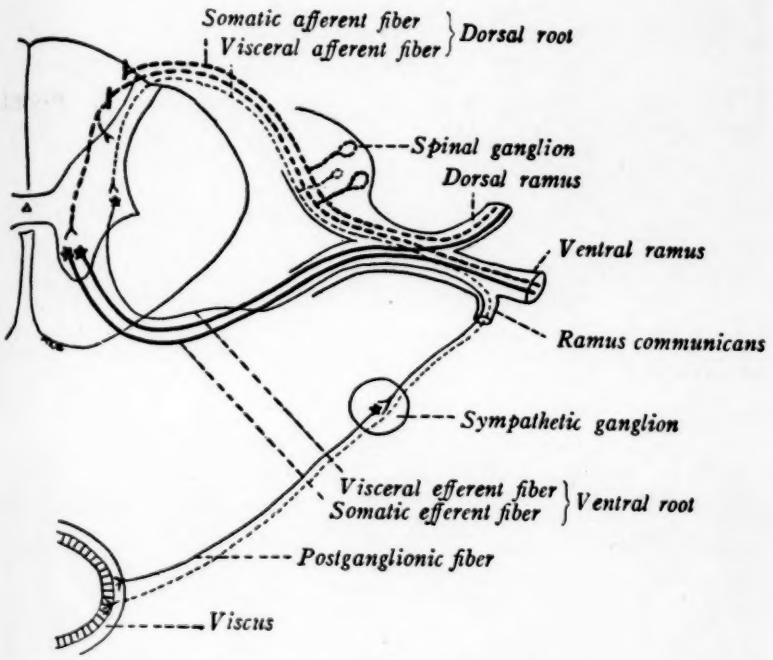

FIG. 1.—Diagram to show the anatomical connections of the preganglionic and postganglionic neurones of the autonomic nervous system. (Ranson.)

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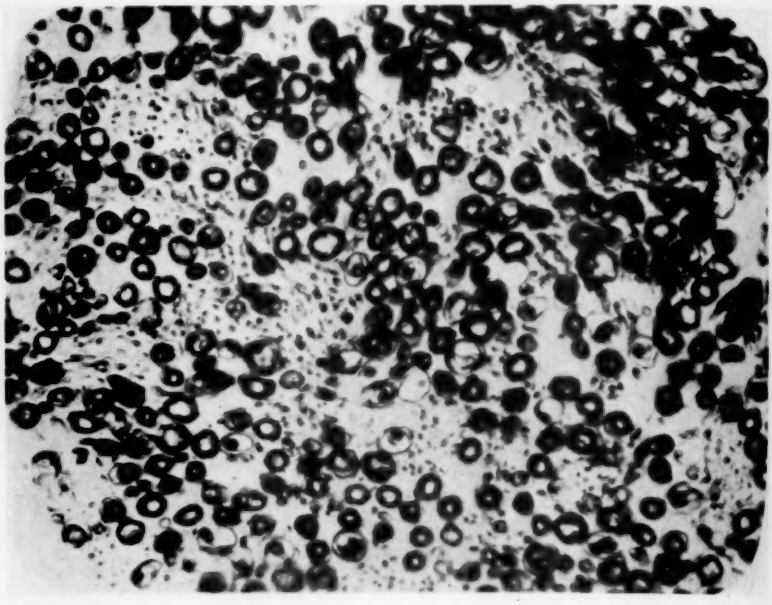

FIG. 2.—Medium magnification of ventral root of fifth thoracic nerve, human, Weigert stain. This shows the intermingling of small-calibre connector fibres with the large medullated motor fibres.

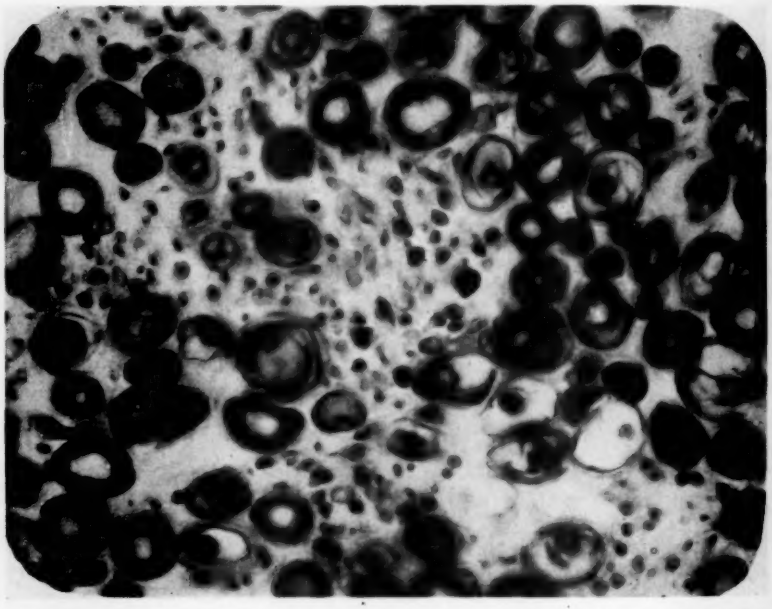

FIG. 3.—High power magnification of ventral root of fifth thoracic nerve, human, Weigert stain, to show the difference in size between the small medullated connector fibres and the large medullated motor fibres.

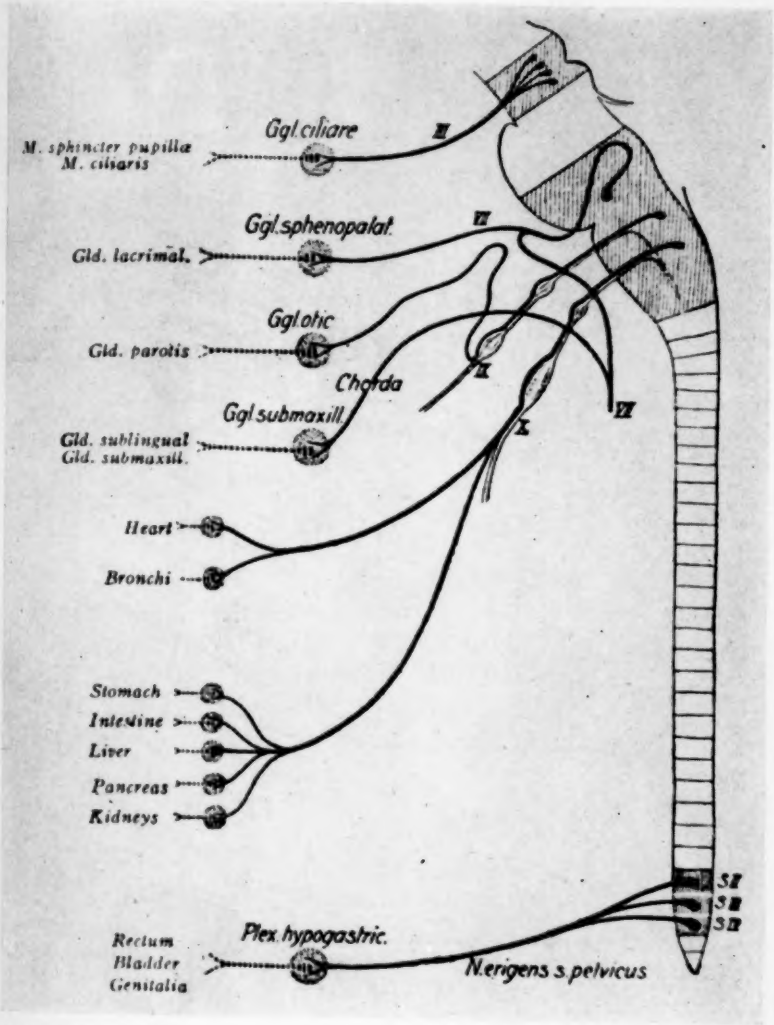

FIG. 4.—Schematic representation of distribution of cranial and sacral (parasympathetic) outflows of connector fibres. (Villiger.)

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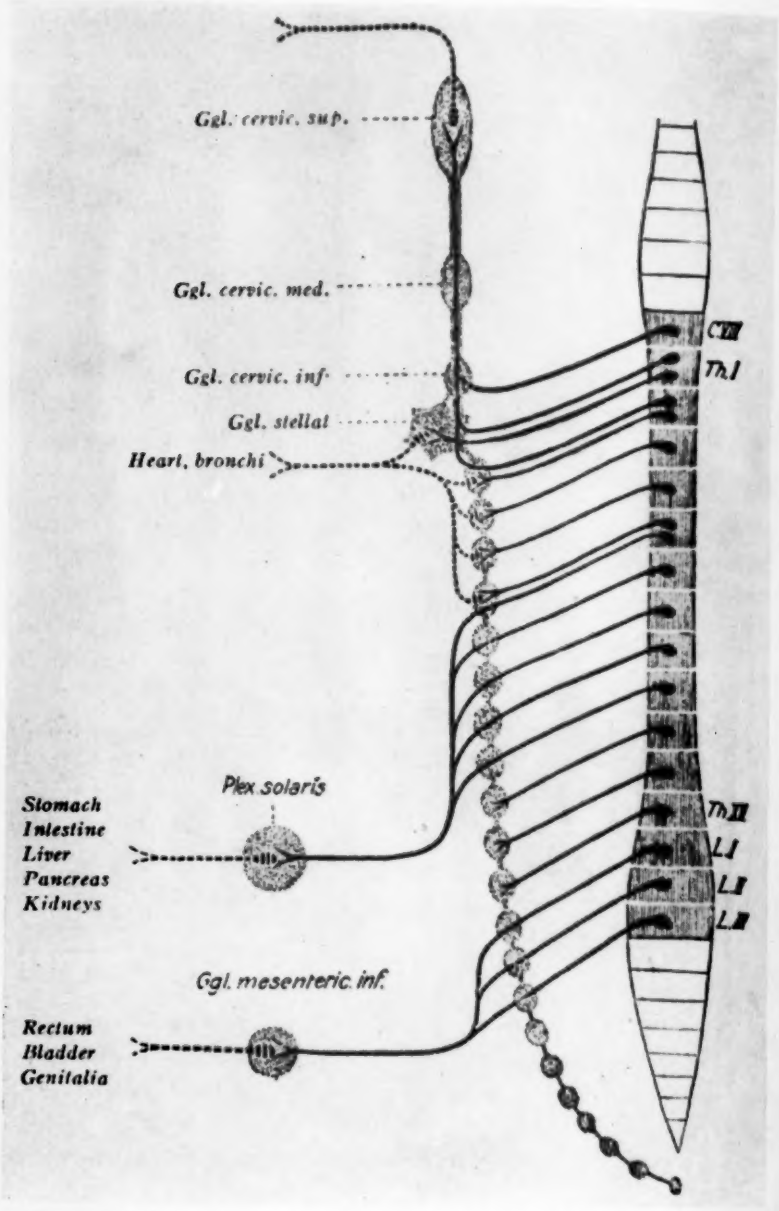

FIG. 5.—Schematic representation of distribution of thoracicolumbar (sympathetic) outflow of connector fibres. (Villiger.)

Langley and Dickinson [2] followed Gaskell and by a series of experimental investigations on cats worked out the position where the preganglionic or connector fibre synapses with its autonomic nerve-cell. They showed that the connector fibre of the thoracico-lumbar outflow had, as a rule, its synapse with a nerve-cell in one of the paravertebral autonomic ganglia, whereas the synapse of the cranial and sacral connector fibres tended to be in the pre-vertebral or terminal autonomic ganglia. Their researches suggested that, as the autonomic nerve-cells connected with the para-sympathetic outflows were nearer the periphery (in the prever-tebral or terminal ganglia), it was probable that this system was earlier in phylogenetic development than the sympathetic division, the autonomic ganglion cells of which had only wandered to the periphery as far as the paravertebral ganglia. It seems likely, therefore, that the sympathetic or katabolic division of the auto-nomic system is a later development in the animal series than the parasympathetic or anabolic division. Langley and Anderson were also responsible for the first use of the universally accepted terms of sympathetic and parasympathetic as applied to the two divisions of the autonomic nervous system.

FUNCTIONS.

One of the first important generalizations bearing on the func-tions of the autonomic nervous system was made by Eppinger and Hess [3] in 1909, when they suggested that individuals tended to fall into two groups, the vago-tonic and the sympathetico-tonic. The former type is represented by the stolid, apparently unim-aginative person, who puts on a reasonable amount of weight, shows a slow pulse, a low metabolic rate and a cheerful, unworried outlook on life. Such a person they believed to be the possessor of a dominant para-sympathetic division of his autonomic system. The antithesis of such an individual, the sympathetico-tonic person, is irritable, " highly-strung," full of energy, thin, pushing and gen-erally objectionable. Such a type is seen to an exaggerated degree in hyperthyroidism, where an overactivity of the sympathetic divi-sion of the autonomic nervous system is considered to be an accom-paniment of the pathological state. The average normal person shows a merciful balance of action of his parasympathetic and sympathetic autonomic divisions.

Cannon [4] published in 1915 his classical work "Bodily Changes in the Emotions," in which he showed that emotions such as anger and fear caused stimulation of the sympathetic nervous system to overaction, resulting in diffuse katabolic changes in the animal's body evidenced by fight or flight as physiological responses to anger or fear. He therefore confirmed Gaskell's earlier suggestion of the katabolic action of the thoracico-lumbar outflow of connector fibres. This violent response has been evolved to meet and deal with an emergency, and is of necessarily short duration. The capability of reaction to an emotion by fight or flight is developed comparatively late in evolutionary history, and this lends weight to Langley's suggestion that the sympathetic nervous system was evolved later than the parasympathetic system.

Cannon stated further, as results of his physiological investigations, that the cranial outflow is conservative and anabolic in its action and that the functions of the sacral portion of the parasympathetic system are generation and evacuation.

In 1930, the same investigator [5] published his results of complete removal of the sympathetic nervous system in cats. The animals which survived the multiple operations necessary, showed no interference with their vegetative functions, but were unable to react effectively to noxious stimuli and they would therefore be quite incapable of "making their way in the world" outside the protective wire-netting of their cages.

Although the various portions of the parasympathetic nervous system seem to have capabilities of individual, independent action, the sympathetic system generally acts as a whole, showing a diffuse and violent response. It would seem reasonable, therefore, to expect to find centres somewhere in the brain, the functions of which would be the central control of action of the sympathetic and, perhaps, of the parasympathetic division of the autonomic nervous system. Considerable evidence has accumulated that such centres are present in the hypothalamic area of the diencephalon.

The Hypothalamus.

Evidence for a sympathetic-controlling centre.

To Karplus and Kreidl [6] of Vienna should probably go the credit of being the first investigators to produce clear-cut experimental evidence of a sympathetic centre in the hypothalamus. In

1909 they published their results of electrical stimulation of this region, their animals uniformly showing sweating, dilatation of the pupil and a rise in blood pressure.

This pioneer investigation was followed by experimental evidence suggesting the presence, in this area, of centres for sugar regulation, insulin production, adrenalin secretion and heat regulation. With reference to the last-named, Magnus [7] noted that animals which were decerebrated by the standard method of a section through the mid-brain died rapidly of cold at ordinary laboratory temperatures, whereas a decerebration which removed the cerebral hemispheres alone, leaving the thalamus still in connection with the brain-stem, resulted in an animal capable of maintaining a constant temperature. This strongly suggested a heat regulating mechanism in the thalamus or subthalamic area, and a recently published article by Bazett, Alpers and Erb [8] shows that the thalamus can be largely removed, with the maintenance of a constant temperature level.

In 1929, Bard,[9] working in Boston, noted that "thalamic" animals showed various phenomena which he called "sham-rage." A cat, which was previously quite docile and friendly, would show, after decerebration above the thalami, raising of the hairs, arching of the back, spitting and dilatation of the pupils as a response to the most friendly of advances. These evidences of anger suggested to the observer an uncontrolled action of the sympathetic division of the autonomic system. Fulton and Ingraham [10] showed later that complete decerebration was not necessary to produce sham-rage, and that this phenomenon could be seen after section bilaterally of a bundle of fibres which passes from the cortex of the frontal lobe to the hypothalamic region. It seems likely therefore that the sympathetic centre in the hypothalamus is normally controlled by a tract leading from the cortex of the frontal lobe of the cerebral hemisphere.

The more exact anatomical limits and connections of this hypothalamic centre were worked out by Beattie, Brow and Long [11] of McGill University, who published their experimental results in 1930. They based their work on a previously noted observation that a normal cat, when anæsthetized with chloroform, shows extrasystoles in its heart beat. These extrasystoles are not observed during chloroform narcosis, if the splanchnic nerves have been pre-

viously cut and the adrenal glands removed. For these reasons the extra-systoles were considered to be an index of sympathetic stimulation. The McGill observers noted that the extrasystoles stopped immediately following transverse section of the mid-brain at the level of the superior colliculus. They therefore argued that there must be a sympathetic-controlling mechanism above this point. Following a section of the brain stem passing from the superior colliculus dorsally to the posterior aspect of the optic chiasma ventrally (Fig. 6), the extrasystoles were found to continue without interruption and hence these workers localized the sympathetic centre in a series of three ganglia which lie between these two lines of section, and which have been called collectively the posterior hypothalamic nuclei. These observers were further able to keep cats alive for short periods after transection of the mid-brain at the level of the superior colliculus, and to trace a tract of degenerating nerve-fibres from the cells of the posterior hypothalamic nuclei, through the brain stem and spinal cord, to their synapses round the nerve-cells in the lateral horn of grey matter of the thoracic spinal cord, which transmit the sympathetic nerve-impulses to the heart (Fig. 7).

There is considerable anatomical evidence, therefore, not only that these posterior hypothalamic nuclei contain cells controlling the sympathetic mechanism, but that the axones of these cells form an anatomical nerve-tract, which connects them with the spinal cord cells mediating the sympathetic nerve-impulses which travel to the viscera.

The following clinical case may be cited as an example of a pathological lesion stimulating the sugar-regulating centre in the posterior hypothalamic region:

A woman of 63 fell unconscious while working in her garden, and died two days later. Examination showed large quantities of sugar and albumen in the urine with .29 per cent of blood sugar. She was not considered clinically to be a case of diabetic coma as the ketones in her urine showed only an insignificant increase over normal. At post mortem intracranial hæmorrhage was found to have occurred from rupture of an aneurysm of the middle cerebral artery. Two small hæmorrhages were found in the brain stem, one in the position of the posterior hypothalamic nuclei and the other on the course of the hypothalamo-spinal tract. (Fig. 8.)

The probable presence of a centre in the posterior hypothalamus, which is, in its turn, probably controlled by nerve-cells in the

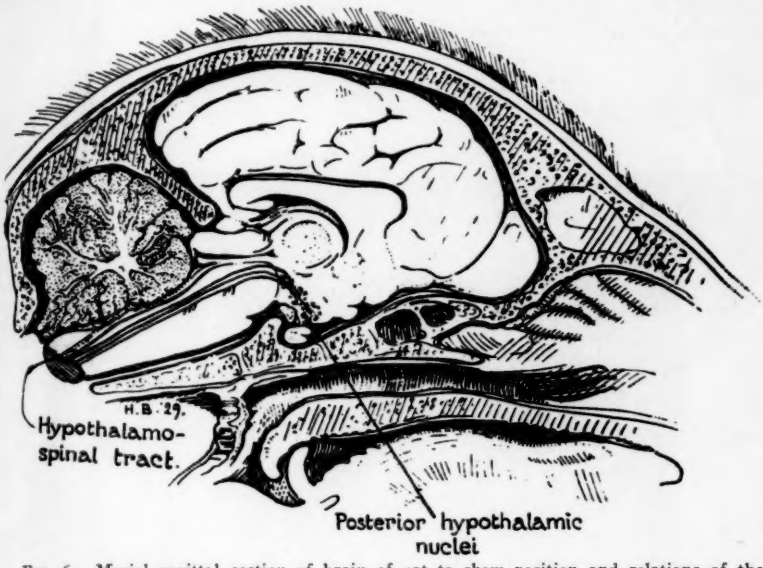

FIG. 6.—Mesial sagittal section of brain of cat to show position and relations of the posterior hypothalamic nuclei. (Beattie.)

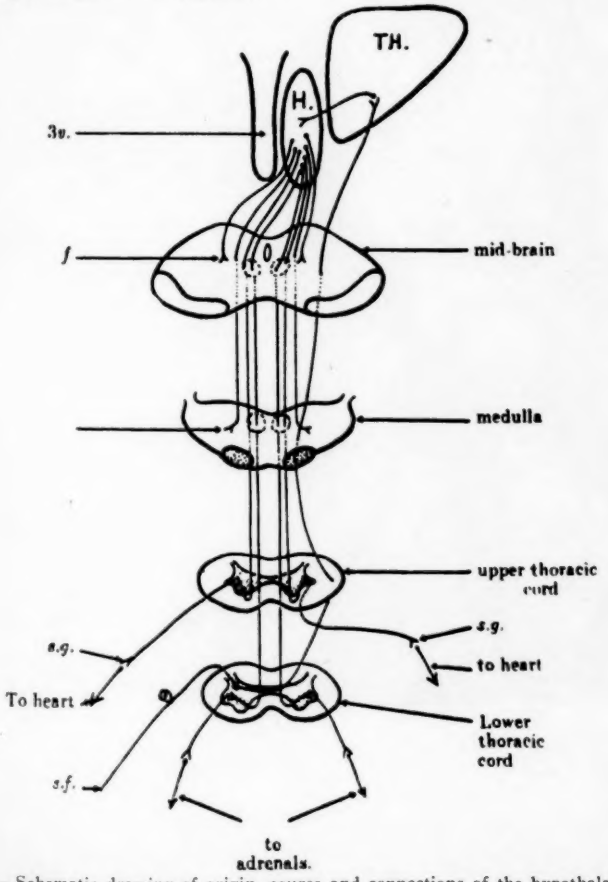

FIG. 7.—Schematic drawing of origin, course and connections of the hypothalamo-spinal tract. (Beattie.)

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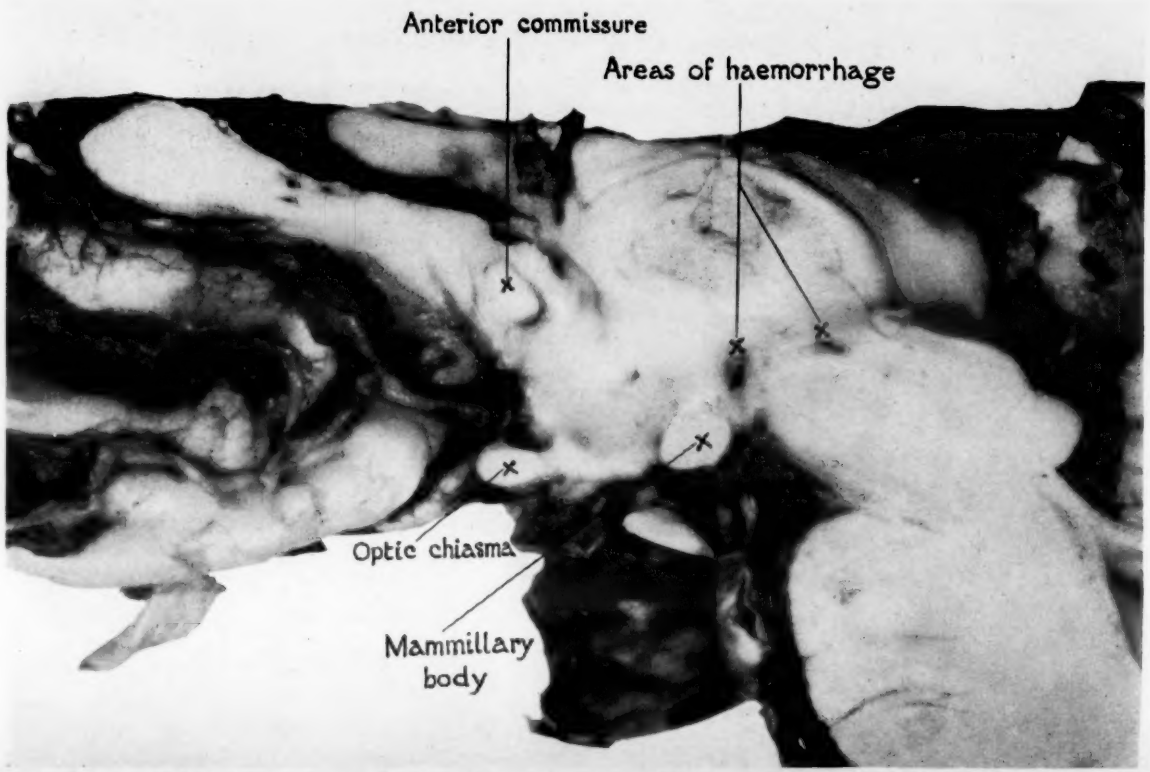

FIG. 8.—Sagittal section of human brain showing hæmorrhage in the regions of, (1) the posterior hypothalamic nuclei and, (2) in the position of the hypothalamo spinal tract.

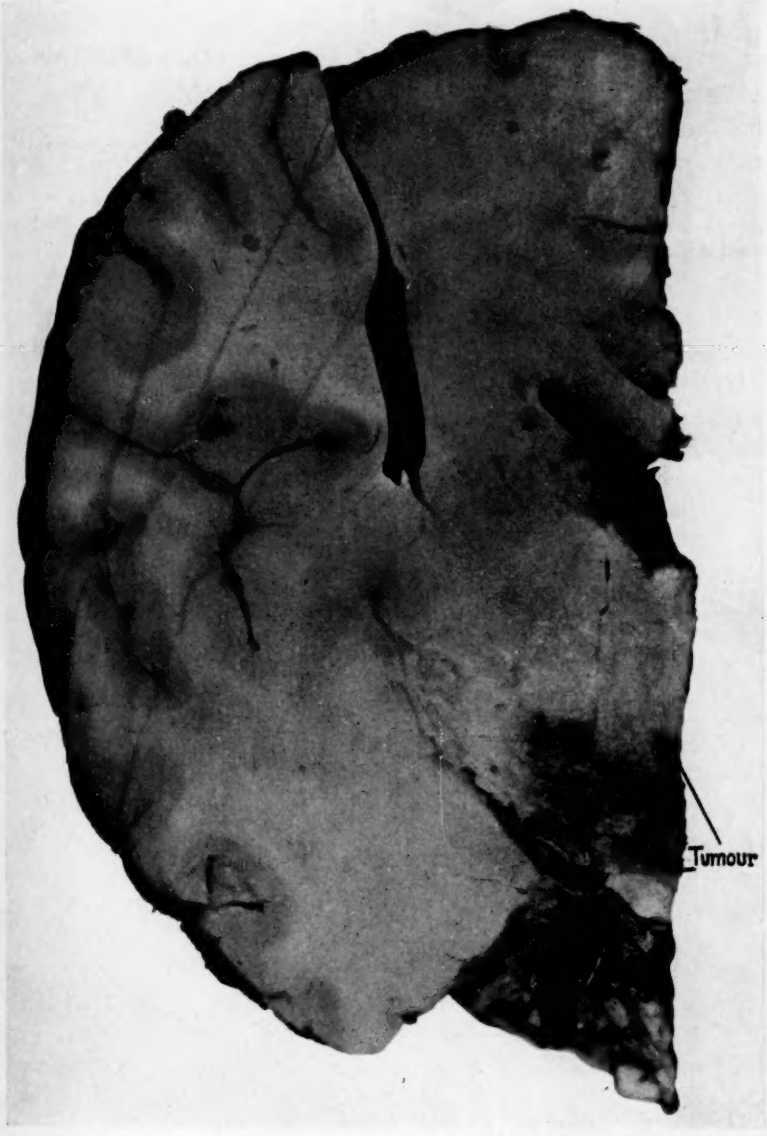

FIG. 9.—Vertical section of right hemisphere of human brain cut immediately behind the optic chiasma to show position of glioma (spongioblastoma multiforme) invading the hypothalamic tissue containing the tuberal group of nuclei. This tumour gave symptoms suggestive of encephalitis lethargica.

FIG. 8.—Sagittal section of human brain showing hæmorrhage in the regions of, (1) the posterior hypothalamic nuclei and, (2) in the position of the hypothalamo spinal tract.

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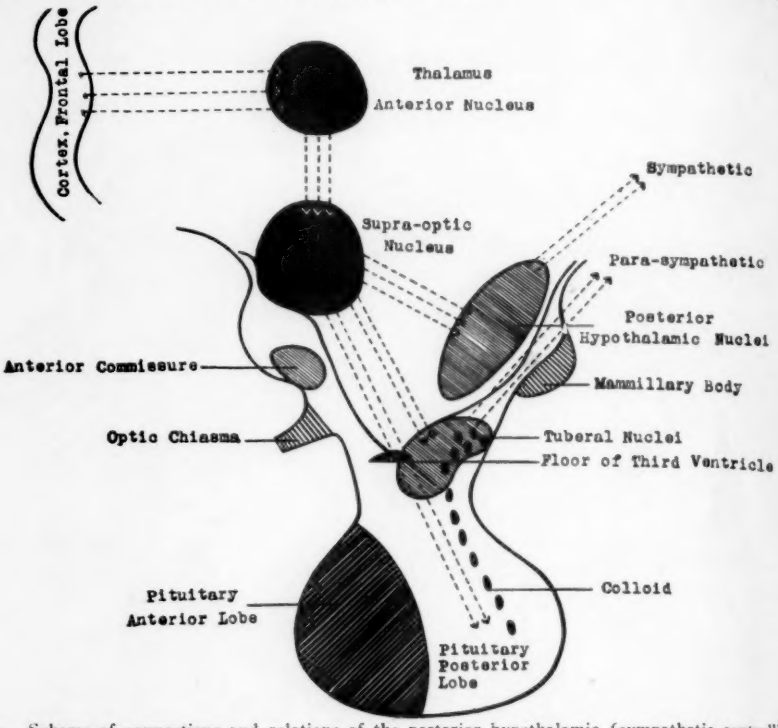

FIG. 10.—Scheme of connections and relations of the posterior hypothalamic (sympathetic controlling) nu and the tuberal (parasympathetic controlling) nuclei. (Beattie.)

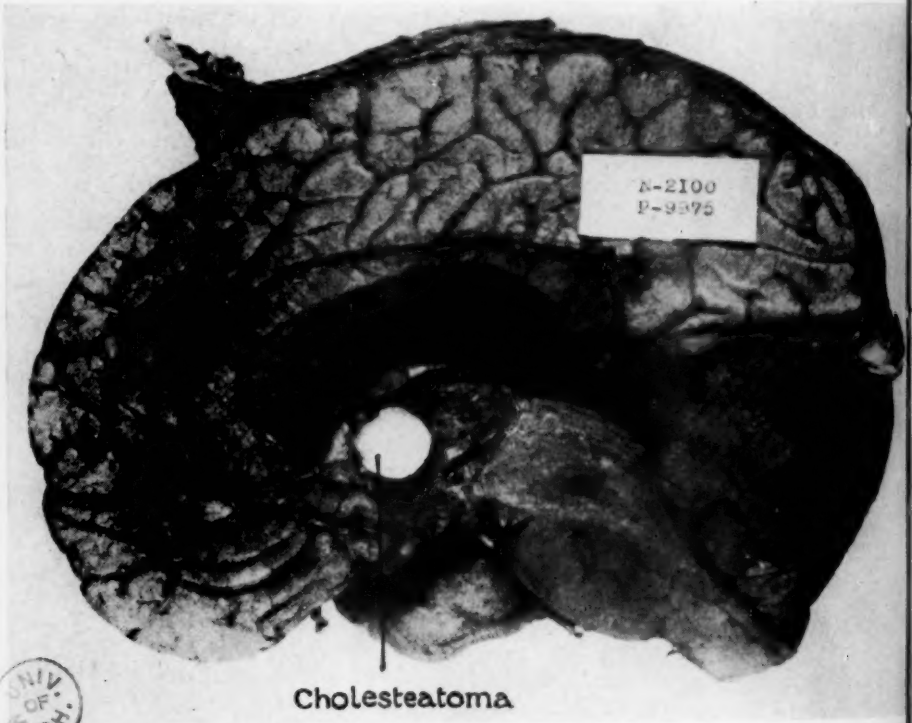

FIG. 11.—A sagittal section of human brain showing a rounded cholesteatoma compressing the ante nucleus of the right thalamus and giving rise to the symptoms described in the text. (Penfield.)

frontal lobe cortex raises many fascinating speculations in the clinical field. In the first place the attacks of rage and other emotional disturbances seen in young children are gradually suppressed by training of the child. Translated into anatomical and physiological terms, this may mean the gradual acquirement of cortical control over the automatically-acting sympathetic hypothalamic centre. Again, the general state of a patient suffering from "shell-shock" suggests an individual whose sympathetic system is over-active, and who has many of the physical attributes of hyperthyroidism. It might be suggested that such a person had a constitutionally weak cortical control of his hypothalamus and that the shock of the shell explosion had further weakened this control with, as a result, the appearance of the phenomena of "hypothalamic release." Lastly, many cases of mental derangement with cortical degenerative changes, as for instance general paresis, must suggest to the observer loss of cortical control over lower emotional centres.

Evidence for a Para-Sympathetic Controlling Centre.

Perhaps the first experimental evidence suggesting that the anabolic, vegetative, parasympathetic division of the autonomic nervous system might be controlled by centres in the hypothalamus, was that of Hess.[12] This observer has been able to tie electrodes, with their terminals in the hypothalamus, into cats. After recovery of the animal from the operation, the passage of an electric current through these electrodes has put the cat into a condition which, the observer states, is indistinguishable from normal sleep. This investigator had not only been able to perform this amazing experiment repeatedly, but has been able to take a moving-picture film, in which his results are clearly shown.

The hypersomnia which accompanies encephalitis lethargica is therefore explicable on the assumption of a "sleep-centre" in the hypothalamus, which is stimulated by the inflammatory process. It is, of course, generally accepted that the maximum pathological lesions found in this disease are in the hypothalamic nuclei and in the upper part of the mid-brain.

That drowsiness may be the main symptom pointing to a lesion in this area is clearly shown in the following case.

A farmer of 47 gave a history of lack of initiative and ambition for six months. During this time he had noted gradually increasing drowsiness so

that eventually he would fall asleep while at work, driving his plough. A fortnight before his death he developed severe pain in the back of his neck and head and his temperature was elevated to 102°. He became mentally confused, with unintelligible speech a week before death. On admission to hospital, on the day before death, he was vomiting. His optic discs were examined and showed a doubtful amount of choking. A clinical diagnosis of encephalitis lethargica was made with some confidence. Post mortem examination revealed a malignant glioma (spongioblastoma multiforme) in the hypothalamus on the right side (Fig. 9). This tumour was situated anatomically immediately behind the optic chiasma and was invading the floor of the third ventricle in this situation. There was no evidence of inflammation in the cerebral tissues. The area of hypothalamus involved in this tumour growth is, therefore, definitely anterior to the position of the posterior hypothalamic nuclei. (Fig. 6.)

The next important evidence of the presence of hypothalamic centres controlling vegetative functions was presented in a series of papers by Cushing [13] published in 1931. He injected pituitrin and pilocarpine into the ventricles of patients and obtained, as a result, the phenomena of vasodilatation, sweating, lacrimation, salivation and vomiting. These effects, pointing to parasympathetic stimulation, could only be produced if the drug was not mechanically prevented from being absorbed by the ependyma lining the cavity of third ventricle. The effects were also abolished by the injection of atropin, which paralyses the peripheral terminations of the parasympathetic system. Avertin is believed to exert its anæsthetic action on the hypothalamus, and Cushing was able to show that pituitrin injected into the ventricles was unable to exert its parasympathetic stimulating effects in a patient under avertin anæsthesia. He therefore suggested that there were groups of nerve-cells lying under the lateral walls and in the floor of the third ventricle, stimulation of which was capable of producing overaction of the cranial division of the parasympathetic nervous system.

In the following year, 1932, the same investigator [14] showed that the parasympathetic stimulation induced by tumours involving the walls of the third ventricle might be sufficiently severe to cause acute ulcers to appear in the œsophagus, stomach or duodenum, and that the immediate cause of death, in certain of these patients, was general peritonitis resulting from perforation of such acute peptic ulcers.

Other disease which affects the lateral wall and floor of the third ventricle may produce similar distant effects, as is shown by the following observation:

A girl of 16 died of tuberculous meningitis after an illness of 19 days' duration. Tubercle bacilli were found in her cerebro-spinal fluid six days before death and for the last four days she had severe vomiting. At post mortem typical tuberculous granulomatous material was found filling the interpeduncular cistern of the subarachnoid space, in contact with the floor of the third ventricle. There was granular ependymitis of the lateral walls and floor of this ventricle. Microscopically, lymphoctyes were seen infiltrating the perivascular sheathes of the vessels supplying the hypothalamus and the nerve-cells of this structure showed degenerative changes. Sub-ependymal tuberculous foci were found in the lateral walls and floor of the third ventricle. Examination of the abdomen showed acute ulcers of the stomach and duodenum. The duodenal ulcer had perforated.

Stimulated by Cushing's observations, Beattie recollected that certain of his experimental animals, in which he had sectioned the hypothalamo-sympathetic tract, had died of general peritonitis. He had attributed this occasional finding to infection, caused by the intraperitoneal injection of anæsthetic solutions, and these animals had not been carefully examined for the possible presence of perforated peptic ulcers. In 1932, this author [15] was able to publish a preliminary report on the effects of electrical stimulation of the base of the stalk of the infundibulum. He found increased peristalsis and secretion of the stomach with, after an hour's stimulation, patches of hyperæmic mucous membrane on the lesser curvature of this organ. He also observed an increase of tone of the urinary bladder and a slowing of the heart rate. He found, microscopically, hæmorrhages into and under the gastric mucous membrane, which in time might have proceeded to the formation of ulcers.

The nerve-cells which are believed to control parasympathetic activity are found in a group of nuclei placed in the tuber cinereum at the point of origin of the infundibulum, and which are therefore called the tuberal nuclei. There is some experimental evidence that the axones of these cells synapse round the cells of the cranial nerve nuclei concerned with the transmission of parasympathetic nervous impulses to the periphery.

It may therefore be considered probable that there are nuclei of nerve-cells in the hypothalamic region which control respectively the sympathetic and the parasympathetic divisions of the autonomic nervous system. The former lie in the posterior hypothalamic region, the latter being placed more anteriorly in the tuber cinereum at the base of the infundibulum. (Fig. 10.)

Penfield [16] published in 1929 the case of a woman who had periodic seizures, the motor and secretory phenomena of which were limited to tissues supplied by the autonomic nervous system. She showed in these attacks a prodromal restlessness, followed by sudden vasodilatation of the skin in the area of supply of the cervical sympathetic and a sudden rise in blood pressure. This was succeeded by lacrimation, diaphoresis, salivation, dilatation (or contraction) of the pupils, increased rate and pressure of the pulse, retardation of respiratory rate, elicitability of the pilomotor reflex and occasional loss of consciousness. The superficial blush then disappeared, with a fall in blood pressure and a slowing and weakening of the pulse. She hiccuped 3 or 4 times, showed transient shivering, and finally, Cheyne-Stokes respiration was noted immediately before she recovered.

These changes were suggestive of stimulation of both the sympathetic and the parasympathetic autonomic divisions. Autopsy showed a rounded cholesteatomatous tumour compressing the anterior nucleus of the right thalamus. (Fig. 11.) Reference to Fig. 10 suggests the theoretical possibility that the nerve-cells of the anterior nucleus of the thalamus may be able to send nervous impulses for the activation of both the posterior hypothalamic and the tuberal groups of nuclei.

Conclusions.

In this review of the structure and functions of the autonomic nervous system, followed by an account of present conceptions of the hypothalamic controlling centres for this system, the author acknowledges how very much "overworked" is this small area of the brain.

It is very probable that many current views will have to be modified in the future, and that some will be abandoned as knowledge increases. The practical importance of the main ideas embodied

in these investigations, and the light which they throw on the hitherto obscure symptomatology of many and varied disease processes in the human subject, are sufficient justification for their statement.

Grateful acknowledgments are made to Drs. Beattie and Penfield for allowing reproduction of some of the illustrations which they have already published. The author also wishes to thank the various members of the Medical Staff of the Toronto General Hospital who have permitted the publication of clinical details of their patients.

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CONCERNING PICK'S DISEASE.*

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In 1892,† Arnold Pick, of Prague, found in a study of senile brains that some specimens showed more or less circumscribed areas of cortical atrophy which were most conspicuous in the frontal lobes, but also occurred in the temporal lobes, and, if rarely, elsewhere. Pick, being interested in aphasia, saw the possibility that these pathological findings might be the basis of certain clinical focal manifestations. Studies of the case histories revealed that these patients had shown progressive dementia and rather frequently aphasic disturbances.

Continued pathological and clinical observations brought additional information, namely, that senile plaques and intracellular fibrillary changes are notably absent; that arteriosclerosis plays no rôle in etiology and that in the majority of cases the onset of symptoms is prior to the seventh decade of life. Many investigators have confirmed these findings, especially in pathologico-anatomical respects, and have accepted Pick's disease as a definite clinical and anatomical type which is classified as a presenile psychosis.

Pathologically, there may be a relatively diffuse cortical atrophy, but the circumscribed areas of intense atrophy in certain lobes or even convolutions are distinct and readily discernible with the naked eye. The areas involved are not exactly symmetrical and are usually more marked on the left side. The focal atrophies may be present in the pre-fontal lobes (including Broca's area), the temporal lobes, and less frequently or less intensely in the parietal and occipital lobes and the basal ganglia.‡ These focal areas may

* Read at the eighty-ninth annual meeting of The American Psychiatric Association, Boston, Mass., May 29–June 2, 1933.

† This date is quoted from a paper published by Pick in 1898. (See bibliography.)

‡ Braunmühl has demonstrated primary and characteristic changes in the globus pallidus and substantia nigra. He mentions such a case which showed hyperkinetic symptoms and very little dementia or asphasia. Hoedemaker and Mathews state that the caudate nucleus and the anterior and lateral thalamic nuclei may be involved.

present themselves in numerous combinations, following no definite law, but the motor cortex and the cerebellum remain essentially free from disturbance. Spatz has pointed out that the disease affects the phylogenetically younger parts of the brain.

Histologically, degeneration and destruction of ganglion cells are noted in the first three cortical layers and to a less degree in layers five and six. The neuroglia structure shows compensatory hypertrophy and regressive changes. As stated above senile plaques and Alzheimer's fibril changes are not found, and cerebral arteriosclerosis is absent or only very slight.

Although a very definite and characteristic anatomico-pathological basis for Pick's disease has been established, the clinical picture has not assumed quite such definite form, and it is hoped that the presentation of clinical observations will serve to create interest in increasing our knowledge of the clinical symptomatology of this condition. Close study of the symptomatology brings one the conviction that detailed observations of the evolution of the disease portray characteristics and sequences of development that aid materially in building up a clinical picture that will in many instances differentiate Pick's disease from similar conditions such as Alzheimer's disease.

Generally speaking, two types of Pick's disease have been described—one characterized by dementia and aphasia and the other by dementia alone. The anatomical basis for these two possibilities is obvious. Carl Schneider has also pointed out that there may be acute and chronic forms.

In the beginning of the disease, as in some other organic disturbances of the frontal lobes,* such as paresis, the more complicated social adjustments, the higher or later cultural and ethical standards and the finest spiritual accomplishments are the first to break down, leaving formal memory intact for considerable time. Usually before gross memory defect is demonstrable, attention is less easily gained, giving the impression of indifference or sup-

* The case of Pick's disease which was described when this paper was read was originally diagnosed as a frontal lobe case in May, 1930. Later symptoms of temporal lobe and basal ganglia involvement were noted and in October, 1933, the post mortem examination confirmed the diagnosis. Complete details of this case, including the studies of the neuropathologist and the psychologist will be published at a later date.

pressed emotion and reasoning ability becomes impoverished and stereotyped. Apparently memory *per se* is impaired very slowly, but the use of memory (as an intellectual tool) becomes handicapped early. That memory material is still preserved can be seen in rather progressed cases. One may get the impression that the patient is unable to find access * to his memory. (Contrarily, in Alzheimer's disease, the memory itself is destroyed.)

As the disease progresses, partial disorientation occurs, emotional lability increases and a rather characteristic stage of hyperactivity ensues.

In this period of heightened activity, because of the early blunting of social and ethical qualities, the patients may give up their positions; indulge in questionable drinking or sex activities; squander their money; lie and even steal, and for the first time the relatives or friends begin to be suspicious. A little later the patients become more childlike; wander about, sometimes getting lost; tell poor jokes and keep repeating the same stories over and over. If brought to a hospital at this time they busy themselves about their rooms, cleaning and rearranging things, and their activity becomes less and less purposeful, sometimes with a repetition of rather bizarre acts. Even when sitting in a chair they may continue such stereotyped movements as tapping the foot on the floor or turning page after page of a magazine. They often keep themselves busy far into the night.

Through this stage of hyperactivity, the memory becomes less and less accurate, but not as grossly impaired as it appears to be at first sight, the patients being able at certain times to recall details with surprising facility. The attention defect with the general indifference may give the observer the impression of quite widespread memory defect. The attention disturbance in itself is complicated, but may in some instances appear to be increased by the patients' realization of their handicaps with the resulting effort to conceal their difficulty by avoiding contact.

If during this hyperactive stage, temporal lobe atrophy complicates the disease, the patients begin to show aphasic symptoms. Sensory aphasia, producing difficulty in understanding the spoken or even written word may increase the attention disturbance,

* Frontal lobe atrophy producing lack of initiative may account for this.

thereby giving what appears to be an augmentation of memory defect for recent events. Likewise, motor aphasia, by causing difficulty and embarrassment in expression and consequently withdrawal from contacts may produce a train of secondary symptoms. All varieties, combinations and degrees of aphasic disturbances may be found and sometimes in addition apractic symptoms. Vocabulary and speech diminish but there is no dysarthria or logorrhea. Echolalia and perseveration occur frequently.

Gradually with the progress of the disease, the activity subsides, speech disappears; orientation and memory become more impaired; emotional responses become greatly diminished; initiative is lacking and the patients settle into a state of hebetude.

In this semi-final stage, characterized by inertia, the patients would sit or lie all day if not activated by rather strong stimulation. They live a vegetative existence while the distinguishing qualities of their former personalities are blotted out. Rarely will a voluntary act appear except where some old well-established habit such as eating, eliminating or sleeping is concerned, and even here the lack of initiative increases until the patients have to be fed and are incontinent. Some simple stereotyped movements such as patting the arm of the chair may continue for some time when once initiated.

A definite resistance to all efforts to stir them develops, and this resistance gives the impression that there is a generalized rigidity of the body. When the patients' hands grasp an object there is a tendency to hold on. This may be part of the general resistiveness; it may be related to the grasping reflex or we may think in this connection of the possibility of brain stem involvement. In posture, the patients become stooped and the arms tend to hang forward with slight flexion, suggesting somewhat the posture of the great apes. Gait is naturally slow and uncertain with short steps and finally the patients are no longer able to walk.

When speech disappears entirely it becomes difficult to estimate the memory disturbance but special tests will show that here and there at various times there is still some retention of remote and even recent impressions.

Aphasic disturbances are likewise difficult to estimate at this stage. Because of the extreme inertia and loss of speech it may appear that the patients do not understand or on the other hand a complete auditory agnosia may be responsible for the lack of

response. The same is true in regard to other manifestations of aphasic disturbances. However, it may be possible to show that a patient can still respond in writing or even understand written words, and the writing may show repetitions or perseveration. If a patient is first seen when this stage is well developed, the differentiation between the frontal and temporal lobe types and also between Pick's disease and Alzheimer's disease is practically impossible from a cross-section view.

This state of hebetude is characterized by indifference to the environment and spontaneous emotional responses are practically nil. Patients often masturbate in the presence of others without any evidence of shame. However, at times a smile or a wrinkling of the forehead may be produced with adequate stimulation. A relaxation or change in facial expression may occur when a meal is presented or something is done for the patient's comfort. A sudden gesture toward the face will bring a defense posture with change in facial expression suggesting fear.

Finally a state of perfect passivity and inertia is reached with incontinence and loss of strength; the patients are continuously bedridden and some intercurrent infection leads to death.

In order to complete the clinical picture some general remarks must be added. The average age of onset is between 50 and 60 years of age, and the duration of the disease may be from two to ten years. It seems to occur more frequently in women. Transient periods of depression and more rarely elation may appear; hallucinatory experiences and delusions may be noted from time to time but they are not constant and none of these symptoms are characteristic of Pick's disease. Epileptiform spells or transient losses of consciousness occur infrequently.*

The general neurological examination is usually negative. Symptoms referable to the motor and sensory system (except for the aphasic disturbances) are very rare. Occasionally slight weakness of facial muscles or other muscles may appear temporarily but with no definite paralysis. Apoplectiform attacks are practically unknown. Muscular rigidity that is sometimes apparent may be due to resistance, but more detailed study of basal ganglia func-

* Krapf maintains that these epileptiform spells are seen only in cases with hypertension which seldom occurs in Pick's disease.

tions may, in some cases, eventually lead to the clinical demonstration of manifestations of the pathology that has been demonstrated in these regions.

The Wassermann reactions of the blood are negative and all examinations of the cerebrospinal fluid including the hydrodynamics give normal findings.

Encephalograms give valuable diagnostic aid fairly early in the disease, showing confluent air spaces, indicating brain atrophy in the frontal and sometimes the temporal regions. Occasionally an internal hydrocephalus may also be demonstrated.

Several theories pertaining to Pick's disease have been suggested, and will be mentioned briefly. At first Pick was inclined to attribute the atrophic changes to vascular disturbances superimposed on the usual senile process, but further observations revealed a minimum of vascular disease and none that could be directly related to the particular areas involved. Other observers (Gans, Spatz, Stertz, Spielmeyer, etc.) have also demonstrated that vascular changes could not be an etiological factor.

Monakoff's theory that involutional changes in the choroid plexus impairs its function as barrier between blood and spinal fluid, leaving the neurones more vulnerable to circulating toxins is interesting in this connection, but pathological studies have shown no consistent changes in the plexus. Furthermore such a theory would not account for the localization and would be more applicable to Alzheimer's disease as has been suggested by Malamud and Löwenberg.

Some writers have suggested that the atrophy follows Flechsig's zones of myelinization; some believe that the involvement corresponds to the cyto-architectonic areas of Brodmann and therefore is probably a heredodegenerative process. Gans suggests that Pick's disease might represent an abiotrophy, a delayed heredofamilial affection. Critchley favors the conception of a precocious aging of neurones and suggests that occasionally a part of the nervous system may be singled out for this change and that Pick's disease, paralysis agitans and the delayed cerebellar atrophies may well be examples of a selective involutional process. Spatz, pointing out the predilection for changes to occur in the phylogenetically younger parts of the brain, also assumes that there might be some hereditary factor similar to that seen in Huntington's disease. Grünthal has

reported instances of the occurrence of the disease in two brothers and also in two generations of a family. Pick, Gans, Braunmühl, Thorpe, Kufs and others have published cases in which there was a family psychopathic taint. In one of our cases, manic-depressive attacks occurred in two other members of the family.* It seems that at present all we can say in regard to etiology is that there is a possibility that hereditary factors may play some rôle.

From the standpoint of comparative anatomy and physiology the experiments of Fulton in removing certain cortical areas from the brains of monkeys are of interest in comparison with the findings in Pick's disease. Fulton's conclusions are as follows:

1. "Simultaneous bilateral destruction of the true frontal area in monkeys has no effect on posture, *i. e.*, does not give rise to hypertonia, and does not cause forced grasping or retardation in motor adjustments ('perseveration'). It does cause a characteristic syndrome, however, marked by greatly increased spontaneous motor activity with an associated augmentation of appetite." (This is suggestive of the early hyperactivity seen in Pick's disease.)

2. "Simultaneous bilateral extirpation of both frontal areas together with the premotor areas caused forced grasping, perseveration, slowness in initiating responses, but no hypertonia, and the postural disturbances which result are primarily those associated with the phenomena of perseveration and forced grasping and groping." (Perseveration, slowness in initiating responses and possibly forced grasping with or without hypertonia are found in the stage of inertia of Pick cases.)

Interesting as these statements are, it must be remembered that experimental results cannot compare in all respects with a disease process.

In regard to differential diagnosis, the neurological and serological findings, the encephalograms, and the clinical history and picture, will usually rule out or confirm such possibilities as general paralysis, arteriosclerotic brain disease or brain tumor, but the differentiation from Alzheimer's disease is a much more difficult problem. No one symptom or finding is characteristic of either disease; therefore diagnosis must be based on the total findings and the shades of variation found within certain specific symptoms.

* We are under the impression that the majority of Pick and Alzheimer cases, at least in our own experience, are more or less outspokenly pyknic.

In the field of memory disturbances may be found some of the most important points for establishing a differentiation. Usually the onset of memory defect is earlier and more widespread in Alzheimer's disease. Later on the memory is practically obliterated in Alzheimer cases whereas in Pick's disease some evidence of retention can be demonstrated until quite late in the disorder. As stated above in the description of Pick's disease, it appears that memory *per se* is impaired slowly, but the use of memory (as an intellectual tool) becomes handicapped early. One may get the impression that the patient does not find access to memory material which may be there, whereas, in Alzheimer's disease the memory itself is destroyed.

Encephalograms give valuable diagnostic aid in the differential diagnosis. Flügel is certainly right in stating that the encephalograms in Alzheimer's disease show broad stripes of air over the whole convexity, while in Pick's disease the air is almost exclusively in the frontal and temporal regions in the shape of a large confluent mass.

Other less important differential points are as follows: Age of onset and duration do not vary greatly although some Alzheimer cases have been reported in which the age at onset was earlier than any reported case of Pick's disease. In the evolution of the disease processes it appears that in Alzheimer's disease the adequate social behavior and composure is preserved a little longer in spite of the early decrease in memory. Also in the history of the progress of the disease, the stage of hyperactivity followed by hebetude may be a little more clear in Pick's disease. Focal signs, convulsions, etc., are more commonly found in Alzheimer's disease. Recurrent utterances sometimes heard in early Pick cases are rare in Alzheimer's disease, but on the other hand, spontaneous speech, logoclonia and confabulation are all more characteristic of Alzheimer's disease.

In general it may be stated that throughout the courses of these disorders Pick's disease may remind one of general paralysis (excepting the neurological and serological findings) while Alzheimer's disease is more analogous to senile dementia.

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THE CEREBRAL CIRCULATION.

XXVI. Cerebral Anemia—A Discussion of the Mechanism and a Case Report.*

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Two statements about the cerebral circulation found in most textbooks have been contraverted by recent investigations. These statements are: (1) That the cerebral arteries do not anastomose one with another, *i. e.*, they are "end arteries" and (2) that there is no vasomotor control of cerebral vessels, *i. e.*, all their reactions are passive and controlled by systemic blood-pressure changes at a distance.

Fay[1] showed by injections of mercury into the cerebral arteries that the large arteries connect with each other, and that there are even anastomoses between the arteries of the two cerebral hemispheres. The cerebral circulation seems to be designed to avoid local anemia, first, by wide distribution of blood through the Circle of Willis (Fig. 1), then by anastomosis of the larger arteries, and finally by a great continuous capillary bed (Fig. 2). In this capillary bed it has been said[2, 3] that a red blood cell could travel freely from the calcarine cortex to the frontal. This bed is richest about the nerve cells; in the cerebral cortex it is the third, fourth and fifth layers of the gray matter that have the richest capillary network, while the white matter is relatively poor in capillary supply. So it appears that the blood supply is rich in the active gray matter, and poor in the white matter where conduction of nerve impulses is the only function. This difference in blood supply is also apparent in the gray nuclei of the brain stem when compared to the white tracts lying nearby.

Stöhr,[4] Hassin,[5] Penfield,[6] and others have recently shown that there are nerves and nerve endings on the cerebral blood vessels.

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That these have a vasomotor effect has been proved by Forbes and his co-workers,[7, 8]: stimulation of the sympathetic nerve in the neck causes vasoconstriction of vessels in the pia, and stimulation of the vagus nerve in the neck causes vasodilatation. Thus these vessels, as in other parts of the body, have an opposed and balanced innervation: sympathetic *vs.* parasympathetic.

The cerebral vasometer mechanism, however, appears to be much less active than that in other organs; thus when adrenalin is given intravenously the rise in systemic blood-pressure overcomes the slight local constrictor effect on the cerebral vessels, and the blood-flow through the brain is increased.[9] Also the vasomotor effect of stimulation of the sympathetic and vagus nerves is slight and may have little normal functional significance, though it is definite under experimental conditions. It is perfectly reasonable to suppose, however, that pathological changes may make the vasomotor control of cerebral vessels a potent morbid factor; Spielmeyer [10] and others [11, 12, 13] have long thought that vascular spasm plays a rôle in arteriosclerosis, eclampsia gravidarum, cerebral vascular syphilis, epilepsy, migraine and other related syndromes.

The great richness of the capillary supply to the ganglion cells of the brain indicates that these cells need much oxygen. Experiments [14] have shown that the cerebral nerve cells are probably more sensitive to oxygen lack than any other cells in the body. When the carotid and vertebral arteries in a cat are clamped for short periods (8 to 12 minutes) the cat goes into a convulsion. Histological study of the brains of such cats shows that changes have taken place in the nerve cells; devastated areas are found especially in the third, fourth and fifth layers of the cortex; interestingly enough these are the layers with the greatest capillary blood supply. Thus, in animals, about 10 minutes of such *partial anemia* is a "convulsive dose" and the cerebral nerve cells show a concomitant degeneration. *Complete* anemia for 18 seconds will cause a convulsion in man.[15]

CASE REPORT.

On January 1, 1933, a middle-aged Italian laborer had two convulsions and was brought to the Boston City Hospital and admitted on a medical service. Afterwards, it was learned that he had previously received anti-syphilitic treatment from his own physician. A lumbar puncture on January 9, showed

a spinal fluid under low pressure, with 4 lymphocytes, a protein of 20 and a gold sol of 2233332100. The Kahn reaction was negative. Shortly after this, the patient left the hospital against advice, but was again admitted on January 15, because of a series of convulsions. On arrival at the hospital he was in a stupor. An immediate lumbar puncture showed a cloudy fluid at a pressure of about 20 mm. of water, with 62 cells (97 per cent polymorphs); the protein was 33. The patient had a stiff neck and was having frequent convulsions. He was seen by a consultant from the Neurological Service who asked for transfer to the neurological wards. Examination at this time showed the patient to be in a state of *status epilepticus*. He was stuporous and unresponsive. Attacks occurred about every three to five minutes and lasted from thirty seconds to two minutes. Attacks would begin with a twitching of the right eyelid, then in succession the right face, right arm and right leg would be drawn up and jerk. Sometimes the convulsions would finally become generalized. The convulsive movements were slow and postural at first, but as the seizure proceeded they tended to become clonic. Many attacks were confined to the right side only. Between attacks there was a complete right hemiplegia. There was slight drooping of the right face, moderately stiff neck with Kernig's and Brudzinski's signs bilaterally. Both hands showed Hoffman's reflex. All tendon reflexes were hyperactive, more so on the right. There was bilateral ankle clonus and Babinski's sign. The pupils were equal but slightly irregular, reacting poorly to light. There was profuse sweating of extremities. Course moist rales were heard at both bases; the heart was normal and the temperature was normal. Pulse average 100 to 110, respirations 20 to 25.

I saw the patient on January 16, and made the following observations: The attacks were occurring at intervals of about 5-10 minutes. Between attacks the patient was stuporous and showed marked stiffness of the neck with Kernig's and Brudzinski's signs. The attacks were remarkably similar; beginning in the right face there was a contraction of the corner of the mouth and then a turning of the head and eyes to the right; the face also was drawn to the right; all movements were interrupted and clonic. The right hand and wrist then began to move, the right arm was flexed at the elbow and wrist; then the hand, and later the whole arm jerked; next the right leg was drawn up and the ankle began clonic movements at a slow rate. After this the convulsive movements became general. I made several moving pictures of these attacks and recommended large doses of luminal intravenously and immediate antiluetic treatment. Diagnosis: syphilitic vascular disease of the left cerebral hemisphere, probably with a softening in the left motor area.

The blood showed concentration from dehydration; it was otherwise normal. The urine, except for high specific gravity, was normal. Lumbar puncture was not repeated because the puncture on the Medical Service a few hours before showed a low pressure. The N. P. N. of blood taken shortly before death was 68. The patient was given several intravenous doses of sodium luminal in an attempt to control the convulsions.

The patient died eight hours after admission to our ward.

Autopsy Note.—An autopsy was performed by Dr. Leary who removed only the brain. In the gross there was no observable abnormality. The brain was not unusually soft and there was no apparent increase in intracranial pressure. In the left fronto-parietal region, however, there was possibly a very moderate decrease in resistance to palpation. It could hardly be called softening.

The brain was hardened in formalin and five days later was cut in frontal sections. There was no softening, but on close inspection it was observed that throughout the pre-central and post-central cortical areas there was a diffuse pallor of the gray matter, and the line between the gray matter and the white matter was less distinct than normal (Fig. 3). In these areas there was perhaps a slight reduction of resistance to the palpating finger. Specimens were removed for microscopical study.

Histological examination showed a marked, acute degeneration of the nerve cells in the specimens taken from the left pre-central and post-central areas. There were certain sections in which none of the nerve cells took the stain normally—the whole cortex appearing pale and washed out. In the less affected areas there were numerous pale cells, but not enough to give the whole cortex this look of "erbleichung" as it is called by Spielmeyer.[16] Many of the individual nerve cells had eccentric nuclei and rounded outlines with poorly staining chromatin. Others were homogenously stained and shrunken. There were also many nerve cells that had become mere shadows (Fig. 4). Most of the abnormal nerve cells showed an increase in the number of satellitic glia cells (Figs. 5 and 6). There was a moderate infiltration of the meninges with lymphocytes. In sections from both hemispheres there was a perivascular infiltration with lymphocytes and a few polymorphs and plasma cells. Specimens from the right hemisphere showed abnormality of many nerve cells, but these were scattered and, therefore, gave no picture of "erbleichung." The perivascular reaction and meningeal reaction were as in the left hemisphere. Stains for iron with Turnbull blue were negative.

Diagnosis.—Meningo-vascular syphilis.

DISCUSSION.

The histological findings in this case indicate that partial occlusion of one of the branches of the middle cerebral artery was responsible for the *status epilepticus*. In other words part of the sensori-motor area of the cortex of the left hemisphere was suffering from insufficient blood supply for 15 days, more severe during the last two days of life. How shall one visualize the pathological process in the cerebral cortex?

In Fig. 2, an occlusion of an artery in the arachnoid space, for example at X, would cause a great decrease in the blood supply (and hence oxygen supply) of the gray matter near G. If this area

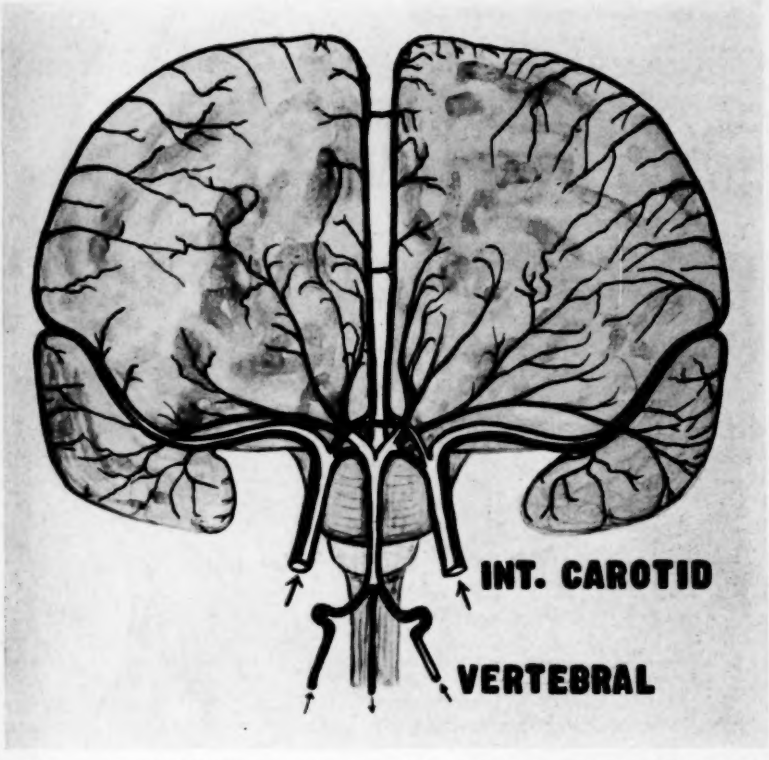

FIG. 1.—Frontal diagrammatic view of the principal cerebral arteries. The vertebral and carotid arteries join to make the Circle of Willis at the base. This anastomotic ring is the "first line of defense" against local cerebral anemia. The three great cerebral branches (anterior, middle and posterior cerebrals) also have numerous connections. There are even connections between the right and left hemispheres. These anastomoses of the artery branches may be called the "second line of defense." For the ultimate joining of all arteries in the great cerebral capillary bed refer to Fig. 2.

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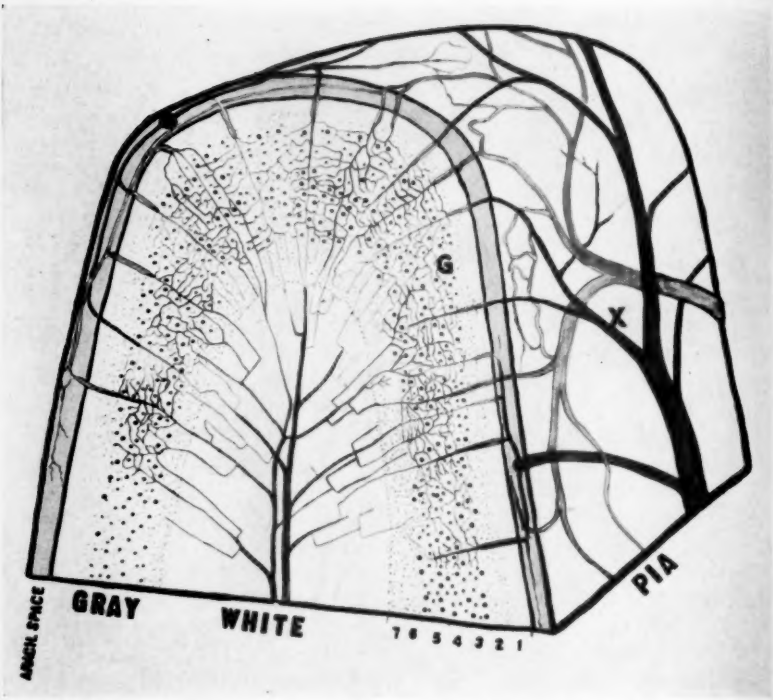

FIG. 2.—Diagrammatic representation of the blood supply of one gyrus of the cerebral cortex. The pial arteries are seen on the surface (black) running in the arachnoid space and here and there sending perforating branches into the brain substance. This is the main source of blood supply to the gray matter, although a few smaller arteries send blood to the gray by way of deep arterioles coming up from the white matter. All these arterioles empty into the great capillary bed, which forms a continuous net all over the cerebral cortex. This is richest in capillaries in the middle layers of the gray matter (laminæ 3, 4, and 5). The venous return is largely outward towards the brain surface, venules taking the blood from the capillary net directly out to pial veins (gray). Note the cuff of arachnoid that dips down into the gray matter, with the small perforating arteries and veins. A minor part of the venous return goes out by way of deep veins in the white matter which join the ventricular venous system and the vein of Galen. (*Cf.* Cobb, Annals of Internal Medicine, 7: 292, Sept., 1933.)

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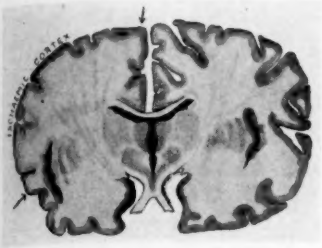

FIG. 3.—Sketch of whole brain section to show location of ischemic necrosis where the gray matter is pale. Blocks for microscopic study were taken from slightly in front of this area (see Figs. 5 and 6), and slightly posterior to it (see Fig. 4).

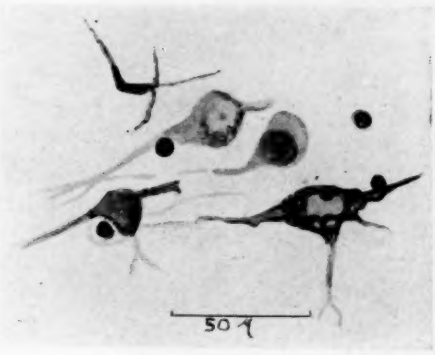

FIG. 4.—Degenerated nerve cells of 6th layer of post-central cerebral cortex. Some are mere shadows. Microglia and oligodendroglia are abundant. (Nissl stain.)

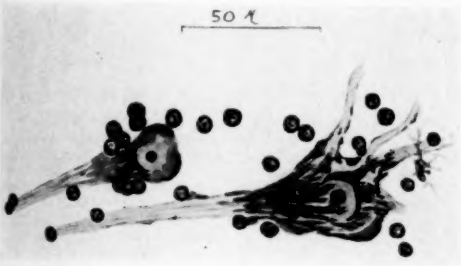

FIG. 5.—The large pyramidal (Betz) cells are undergoing coagulation necrosis; the cell membrane is broken and satellitosis is conspicuous.

FIG. 6.—Small nerve cells from 6th layer of motor cortex. The cells are swollen and chromatolytic; the great satellitosis which extended from the 4th layer to the white matter may have been a factor in obscuring the boundary between white and gray matter.

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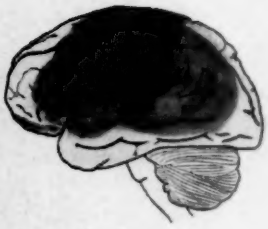
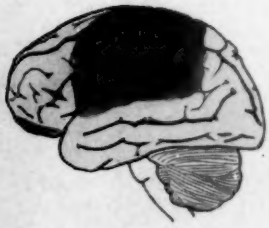
The widespread gray color indicates generalized oxygen reduction of the whole hemisphere, *gradual* in onset; *e. g.*, pneumonia might thus terminate in stupor and death without convulsion.

General widespread reduction of oxygen but of *sudden* onset; *e. g.*, asphyxiation with illuminating gas, coma being initiated by one or more convulsions.



Sudden total occlusion of a large artery, in this case a branch of the middle cerebral, making an infarct of the whole motor area of the left hemisphere. Around this there would be immediately a wide area of edema and partial anoxemia. There might be a convulsion at the onset and the patient would then progress to stupor and death.



Gradual total occlusion of the same large artery: the area of actual infarction and necrosis is equally large, but owing to the gradual onset the outlying area of partial anoxemia is not so wide. The onset here would be more likely to occur with a convulsion because there is more normal cortex remaining in the hemisphere, but the infarct includes the whole motor area, so convulsion in this case would be unlikely.

Sudden occlusion of a small branch of the middle cerebral artery. Here the main infarct lies outside of the motor area, but at this stage the area of partial anoxemia includes the whole motor area. Earlier, the black area representing necrosis was only partially asphyxiated, and the motor area was for a while normal. At that stage the dying nerve cells discharge into the relatively normal motor area, causing repeated convulsions ending in a right hemiplegia; the convulsions stop as the hemiplegia becomes marked.

Illustrates the ideal situation for repeated Jacksonian convulsions: *gradual partial* occlusion of a small branch of the middle cerebral artery, causing a partial anoxemia of the cortex adjacent to the motor area. As long as this area is being asphyxiated, and up to the point of actual necrosis, there will be repeated discharge into the relatively normal, neighboring cortex, resulting in *status epilepticus* of the Jacksonian type.

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FIG. 7.—Diagram of some clinical varieties of cerebral anoxemia, local and general.

is very small, the other arteries and veins supplying this region might maintain the circulation so that no lesion would result, but if the anemic area of brain tissue were large enough, there would be a part of this tissue at the center where inadequate oxygen supply would call forth capillary dilatation, edema and extravasation of cells.[17] This would further shut off the oxygen supply by anastomosing vessels and there would be cerebral softening, at first a "hemorrhagic infarct" because of extravasation of erythrocytes from the collateral blood supply and later an "anemic infarct" when these were absorbed and necrosis took place. Greenfield[18] showed conclusively years ago that this was the process in every infarct; autopsy, of course, can reveal only one or the other type because the process has been stopped by death at a certain stage of development. Nevertheless, the traditional description is still repeated in textbooks.[19]

Such softenings as this are of common occurrence and, as mentioned above, the patient may have an onset of paralysis without any convulsion, but occasionally one or more convulsions usher in the symptoms. One may visualize, however, a condition of relative oxygen lack in cerebral tissue, not severe enough to cause necrosis, but enough to cause the nerve cells to undergo chemical changes; this might make them discharge into the motor area with resulting convulsive muscular contractions. Indeed, it is just such nerve cell changes that have been described by Spielmeyer[10] in patients dying with convulsions. There is no obvious cerebral softening, but the nerve cells have abnormal staining reactions presumably due to partial asphyxia in the cells and resultant chemical changes.

Before accepting as tenable this theory that *status epilepticus* may be due to partial asphyxia of a restricted area of cortex, the experimental and clinical evidence must be reviewed. The physiological experiments quoted above[14] showed that one of the surest and easiest ways to cause convulsions in animals was by sudden, partial cerebral anemia. The histological studies showed focal necrosis, *i. e.*, small scattered areas of neurone devastation in the cerebral cortex.

From the clinical standpoint[22] there is a large group of asphyxial symptoms that must be summarized and discussed. In the first place most deaths are immediately due to lack of cerebral oxygena-

tion, yet few people die in convulsion. In other words, gradual failure of the whole cerebral circulation causes stupor and death without convulsion (Fig. 7, *a*). Sudden failure of the whole oxygen supply to the brain, as in strangulation by tracheal occlusion or heart block,[15] does cause death in a convulsion. The probable explanation of the difference is that in the slow death the whole organism is in a stupor because of toxemia, acidosis or both, and convulsions are inhibited just as if the patient was under the influence of an anesthetic.

The sudden occlusion of a large cerebral artery, for example, the main branch of the middle cerebral (Fig. 7, *c*) by an embolus, causes a hemiplegia, coma and perhaps death, often initiated by a Jacksonian convulsion. When the pathology is thrombosis of the artery the paralysis usually comes on without an initial convulsion. Here again it seems that slow onset and a large cerebral lesion, do something to decrease the irritability of the nerve cells and thus convulsion is avoided. The large infarct of sudden onset probably causes more acute vascular dilatation and extravasation of blood, with greater surrounding edema. The large infarct of slow onset (Fig. 7, *d*) has less of these, but there will probably always be around the central region of actual necrosis a wide zone of partially asphyxiated edematous tissue. It seems reasonable to suppose that if this is widespread the chance of having coma is greater and of having convulsions is less. A somewhat smaller infarct (Fig. 7, *e*) is more likely to cause paralysis initiated by a focal convulsion, because there remains a large area of normal cortex into which the impulses from the partially asphyxiated nerve cells may discharge. If the occlusion of the artery causing such a small infarct is slow, then the conditions are ideal for repeated convulsions (Fig. 7, *f*) for there is a small area of cerebral cortex undergoing partial oxygen deprivation, with the rest of the brain normal enough to respond actively to the shower of impulses from the slowly dying nerve cells. At autopsy no softening is found, only pallor of the cortex, lack of demarcation between white and gray matter, and failure of the nerve cells to stain. This is the "Erbleichung" of Spielmeyer referred to above. Besides explaining many cases of convulsion, it also explains many cases of "hemiplegia without lesion."[19] As in most "idiopathic" diseases, the lesion was there to be seen if one only would look carefully enough and with the proper methods.

Gibbs [20] has recently shown, by means of an ingenious instrument to measure blood flow in the internal jugular vein, that no general reduction in cerebral blood-flow accompanied the onset of the seizure in the 10 patients he tested. His experiments, therefore, prove that few if any epileptic convulsions are initiated by a widespread vasoconstriction in the brain. It remains, however, a perfectly reasonable theory that certain convulsions may be due to small areas of cerebral anemia (due to embolus, thrombosis or vasoconstriction) which act as focal firing points and discharge into other parts of the brain. In fact just such vasoconstrictor spots have been seen during convulsions on the surface of the cerebral cortex of man exposed at operation.[21]

In explaining, therefore, the results of cerebral asphyxia, four main factors have to be known before one can postulate the resulting symptoms. First, the time factor, was the development of the infarct sudden or gradual? Second, the size of the lesion and whether or not some cerebral cortex remained normal near the infarct. Third (and much the same in implication) the state of the general circulation of the patient, and whether or not any cerebral cortical cells were normally functional. Lastly, the location of the lesion, for a lesion affecting the whole Rolandic cortex might eliminate the possibility of repeated convulsion, whereas a large frontal or occipital lesion bordering on the motor area might give just the conditions necessary for *status epilepticus*.

The kind of infarct, therefore, most likely to produce repeated convulsions, may be postulated to be a small area of partially asphyxiated tissue near the motor cortex. Some, at least, of the motor cortex must remain active in order to receive the pathological discharges from the cells undergoing asphyxiation. Probably the occlusion of the artery supplying the asphyxiated area usually is due to gradual thrombosis. (Often syphilitic as in the case here reported.) Thus the necrosis of the nerve cells is a prolonged process and they have opportunity to discharge repeatedly into relatively normal motor cortex.

SUMMARY.

It has long been held that the cerebral arteries do not anastomose, *i. e.*, that they are all "end-arteries"; it has also been held that these arteries have no vasomotor mechanism. Recent investi-

gations have shown that both these ideas must be revised. A case is reported of a patient suffering from local convulsions of the right side, typically "Jacksonian" in character, which resulted in *status epilepticus* and death. Autopsy showed no softening nor any gross lesion of the brain, but the cerebral cortex of the left motor area showed slight pallor. Histologically the nerve cells in this region showed early anemic necrosis. The pathology is discussed, and it is argued that partial anemia is more likely to cause fits than complete anemia; also it is argued that a small focal area of such anemia is more likely to produce *status epilepticus* than a large area.

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DISCUSSION.

DR. SEYMOUR DEWITT LUDLUM (Gladwyne, Pa.).—I had the pleasure of reading Dr. Cobb's paper, and in order that I may limit myself to time, I have jotted down a few points which occurred to me, which were important, it seemed, in the way of discussion.

This paper of Dr. Cobb's is most important. It brings psychiatry to the front line position occupied by physiology today, that is, the capillaries, the lymph channels, tissue spaces and cells.

Dr. Cobb says when the carotid and vertebral arteries are clamped eight to twelve minutes, the cat goes into a convulsion. Thus ten minutes of a partial anemia is a convulsive dose and that partial anemia is more likely to cause fits than complete anemia. The résumé of that idea would seem to be anoxemia.

Now let's compare for a moment the convulsive states which Rowntree showed as occurring in animals subjected to water intoxication and with the postmortem signs of wet brain substance. This would seem to be an opposite point of view to Dr. Cobb's, but I think that it really means the same thing.

My experience with experimental swelling of cells makes me feel that both of these points of view, anoxemia and water intoxication, have the symptom of cell swelling in common. In Cobb's cutting down of oxygen, there must be, in ten-minute doses, for instance, a more acid pH developing by increase of CO_2 because you are cutting down the oxygen. This amount of change in pH can and does produce swelling of cells.

In Rowntree's ingestion of water there is a constant resulting increase in blood wateriness with resulting low specific gravity and viscosity and low protein. In my experiments, those are just the factors that produce swelling of brain cells. So it would seem to me that the one thing necessary in the exhibition of convulsions would be the swelling of cells. In a colloidal sense this can be brought about by change in pH, change in salts, change in concentration, change in lipoids and proteins.

It seems to me that we can get convulsions from swelling, occurring from any one of these factors. In all of them there is developed a low oxygen threshold. We can learn from Cobb about the anemia. We can learn from Cobb and Gibbs about the cerebral blood flow in epilepsy, and we can learn from Barcroft about oxygen deprivations, from others we can learn about calcium, about lipoids and proteins.

There is evidence from many sides and when they are analyzed, it seems that we are dealing simply with different aspects of a colloidal state of the body fluids, that is the pH and salts and proteins and lipoids and the catalysts.

Any one of these factors can alter the consistency of the fluids of the body, produce changes in cells, of which shrinking or swelling are the simplest forms.

In our work at the Philadelphia General Hospital, we uniformly find in fresh cases of epilepsy that we are dealing with body fluids that tend to swell cells introduced into them and the characteristics are in the serum, low specific gravity and viscosity, a low protein, dispersion of the red cells and hydration of the white cells.

I would like to introduce another piece of work along the line of cerebral innervation, some work done by Dr. Rowntree. I introduce it because I think it is interesting in the light of recent demonstration of the double innervation of the vessels.

At Rochester they have released the brain in ten catatonic patients from sympathetic control by alcoholic injections of the last cervical and the first two thoracic sympathetic ganglia, and also by removal of the corresponding ganglion. These procedures resulted in the development of Horner's syndrome, increased temperature of the corresponding parts of the body and lack of sweating. This resulted at best in the complete disappearance of dementia præcox for a month with a recurrence of dementia præcox symptoms in all patients but one. One of these patients developed epilepsy.

I have simply introduced that because it was new evidence to me and has a bearing on this innervation, and I don't think that we can quite feel satisfied with any one factor in the causation of the epilepsy and that we would have to consider the nervous, the innervation, the anatomical and the chemical as well as any degree of each on the other.

HOMOEROTISM AND PARANOIA.*

By A. A. BRILL, Ph. B., M. D.

Very little that is essentially new has been said on the subject of paranoia since Freud gave out his very stimulating work on the subject in 1911.[1] Ferenczi, who was interested in the subject and had previously discussed it with Professor Freud, is the only one who not only confirmed but added a number of very interesting contributions to the problem.[2] I published the first English paper on the subject in 1911, citing a case of paranoia from my hospital practice; it was an exposition and corroboration of Freud's views.[3] From a purely analytic viewpoint there have been but few contributions since then. We can mention Gribelskaja, Hitschmann, and a few others, who wrote about the problem, and later contributions were made by Schilder [4] and Feigenbaum.[5] Hinsie[6] recently reviewed the subject, but fails to mention a very important later work,[7] in which Freud dilated upon the subject of paranoia and showed its relation to delusional jealousy and homosexuality. I consider this a most important contribution not only because it throws much light on the broader aspects of homosexuality and its rôle in normal and abnormal mental life, but also because it evaluates the problem of jealousy as seen in normal love life and in paranoia.

Before Freud homosexuality was considered a pathological phenomenon, an inversion of the sexual instinct, and most sexologists looked upon it as congenital and organic in origin. But, as Freud puts it: "The recognition of the organic factors of homosexuality does not relieve us of the task of studying the psychic processes of its origin." [8] Following Freud's formulations of the sexual instinct,[9] Sadger discussed the psychology of homosexuality,[10] on the basis of early infantile fixations. Those views were later corroborated by a number of investigators, by Coriat [11] and myself [12] in this country. Homosexuality is not an independent phenomenon, but a part of the normal sexual instinct. Like all the other so-called

*Read at the joint meeting of The American Psychiatric Association and the American Psychoanalytic Association, Boston, Mass., May 31, 1933.

perversions, pathological homosexuality may develop on an environmental basis, in my opinion, regardless of its constitutional factors. It has its origin in the polymorphous perversity of the infantile sexuality. Homosexuality is a normal component of the sexual instinct, which in sublimated form plays a useful part in our social relationship. But, if through faulty evolution it becomes accentuated, or inhibited, the resulting disfunction invariably shows itself in the person's social behavior towards both sexes. To express it more precisely, one may have homosexual difficulties of everyday adjustment, to which the sensuous performances of the frank homosexual are absolutely foreign. We must, therefore, repeat that homosexuality should not be viewed in this narrow pathological sense, but as a normal component of the sexual instinct. Like the partial impulses of looking and touching, it may under certain conditions become more or less pathological. Nor must one assume that occasional sexual acts with a person of the same sex necessarily signifies overt homosexuality. The term itself is somewhat ambiguous and I can see why Ferenczi proposed Karsch-Haak's term, homoerotism, as a substitute for the expression, homosexuality. Ferenczi prefers this term because it renders prominent the psychical aspects of the impulse in contrast to the biological term, "sexuality." Ferenczi justly remarks that the designation "homosexuality" is applied to dissimilar and unrelated psychical abnormalities, and that sexual relations with members of a person's own sex may be nothing but a symptom of the most diverse psychic disorders in abnormal and normal life. Thus, homosexuality is very common in early life wherever the sexes are segregated as in boarding schools, but it is given up as soon as opportunities for heterosexual relations are restored. There was a patient in the Central Islip State Hospital, called the "Governor," who was punished by means of an apomorphine hypodermic because he attempted to do fellatio on a young patient. He protested vehemently against the accusation of homosexuality and explained his act as an initiation rite into a secret society. Ferenczi calls attention to the fact that the two kinds of homoerotism—"active" and "passive"—may "belong in the pure cases, at all events,—to quite different syndromes, and that the 'acting' and the 'suffering' homoerotics represent fundamentally different types of men." [18]

In his interesting Schreber case, Freud correlated paranoia and homosexuality by stating that *the delusion of persecution represented a defense reaction against a repressed homosexual wish phantasy*. The hated arch-conspirator is always one (or a surrogate of one) to whom the patient was formerly attached by very strong affectionate ties, but as this love mounted in intensity, it could not be fulfilled. The patient could not accept it because it threatened to sexualize all his feelings and turn him into an overt homosexual; it, therefore, had to be repressed. The past history of the typical paranoiac invariably shows that he was a highly moral person, whose sexual outlets were markedly restricted, often to the point of almost complete desexualization. The history of such a patient shows that either he kept away from women altogether, or made very feeble attempts at heterosexuality. R., whose case I reported, has always been considered very reserved, highly moral, and almost uncommunicative as far as the opposite sex was concerned. It so happened that we were classmates years before he was admitted to my service in the Central Islip State Hospital. I recalled him as a very poor mixer who kept himself aloof from everybody; he had the reputation of an eccentric and quarrelsome person who always assumed a high moral attitude. The only person with whom he had friendly relations was S., another classmate. I was later told by the patient's family that the patient was in no small measure responsible for the fact that S. married his sister. But, very soon after the marriage the patient's attitude towards S. changed. He no longer sought his company and became increasingly critical of him. Within a few months after S.'s marriage, the patient married after a very short courtship, his wife shortly thereafter refusing to live with him because he accused her of infidelity with S., with his older brother, and with other men. When the psychosis fully developed, he made two attempts to kill S., whom he accused as being the leader of a gang of conspirators; he also accused him of immoral relations with his wife, with his mother, and his sister (S.'s wife).

Now, why did this most loved person become his persecutor? How did this reversal of affect take place? These changes were brought about by the ever present ambivalence of feelings coupled with the unfulfillment of his need for love. R. could not become an overt homosexual, so he tried unconsciously to bring about a sexual union between himself and S., through his sister. This

evidently did not satisfy him, and he made another effort in the same direction by marrying a friend of his sister's. When all these efforts could not stem the tide of his attachment to S., he defended himself against the inner perception: "I love him," by the delusion of persecution, which loudly proclaimed: "I do not love him; indeed, I hate him." But, as the mechanism of the paranoid symptom formation necessitates that these inner perceptions should be replaced by perceptions from without, the formula, "I hate him," was changed through projection into "He hates me; hence, I am justified in hating him." The dynamic unconscious feeling, as a result of the outer perception, then resolved itself into the formula, "I really do not love him—I hate him—because he persecutes me." In other words, the patient's effort at hetero- and homosexual object love failed; the libido, narcistically fixated, regressed to the pregenital and anal-sadistic organization; and the destructive or ego impulses were permitted to become dominant. Instead of loving, he resorted to paranoid projection as a defense against homosexual wishes.

But as paranoia is nothing but a distorted homoerotism, let us review briefly the mechanisms that enter into the formation of the latter. In the average case of homosexuality one finds all or most of the following factors: First, there is a mother fixation, which is established very early and which practically excludes other women. This mother attachment then results in identification with the mother, which in a sense enables the son to remain true to her, his first object. This, in turn, favors narcissistic object-choice,[14] which is easier to put into operation than it is to go over to the other sex.* The narcistic fixation in R. showed itself in a typical

* The narcistic fixation may also show itself in lighter disturbances than overt homerotism and paranoia. Many so-called "old bachelors" belong to this class. Here the narcistic fixation results in an incapacity to find a love object sufficiently attractive to marry. Such individuals may lead a weak heterosexual life as far as physical outlet is concerned, but because of their narcistic fixation, they lack that inner unconscious urge which enhances and overestimates the woman, and forces the average man into matrimony. Such bachelors rationalize their inner fixation by disparaging the "modern girl" in comparison to their mothers. The narcism, as such, does not reach, however, the exaggerated height of paranoia, but nevertheless stamps such persons as special types. They are frequently characterized as eccentric, miserly, self-willed, and combative, and some of them show a clear-cut paranoid character.

ego enhancement. He was an important statesman. The President of the United States sought his advice, he was also Ambassador to Spain (delusions of grandeur). He hated S. and wished to kill him because S. impersonated him as the Ambassador and thus robbed him of his high position. S. did this by disguising himself to look exactly like the patient, so that no one could tell the difference between them. This delusion plainly shows that he loved S. because he saw himself in S. The other mechanisms which frequently play a part in the development of homoerotism are: the overvaluation of the penis with the consequent inability to tolerate its absence in the love object; the regard or fear of the father—the last two come under castration; early homosexual seduction; and jealousy of an older brother. Most of these factors played a greater or lesser part in the psychosis of our patient.

The remarkable thing to remember is that both homosexuality and paranoia are based on narcissistic fixation. But, whereas the homosexual reaches the goal of object-choice, albeit on an inverted path, the paranoiac finds it impossible of attainment, and has to regress to ego-libido.

We mentioned the fact that in his feverish endeavor to obtain homoerotic love from S., the patient married his sister's friend, and then accused her of infidelity with S., with his older brother, and with other men. Cases in which jealousy constitutes the whole picture of the psychosis are well known to us; they are often designated as "alcoholic delusional paranoia," despite the fact that alcohol plays no direct part in the psychosis. In our patient the jealousy did not dominate the picture of the psychosis. However, as delusional jealousy is a form of paranoia directly related to homoerotism, I will briefly narrate the history of such a case. It concerns Mr. M., aged 30, who was brought to me by his brother, a physician, because he was extremely jealous of his innocent wife. He questioned her for hours about the smallest details and was convinced that she was unfaithful to him. He was typical of his kind, but he displayed some insight when I told him that his jealousy had no factual basis. He was quite willing to come for analysis, but as he could not refrain from nagging his wife with hundreds of questions about her doings and whereabouts, I separated him from her, and when this, too, did not work, and he made an attempt to kill his father-in-law, I sent him to Vienna to Pro-

fessor Freud. He was there for about 1½ years, and had to return to the United States. He then came to me and I found him improved, but not cured; he still had to question his wife, but had better control of himself, and fully realized that his jealousy had an unconscious background. He returned to Professor Freud, with whom he remained about another year, and when he returned to New York, he felt that he was not well yet. As Professor Freud advised that I continue the analysis, I saw him regularly for about five months, during which he made good progress. When I suggested that he try to get along without my help, I was still in doubt about his future, but I am pleased to say that he has been doing well for over ten years.

According to Freud, jealousy is a normal affective state, like grief. Everybody has occasion to be jealous, and if one does not encounter it in consciousness, "the inference is justified that it has undergone severe repression and consequently plays all the greater part in his unconscious." I cannot do any better than paraphrase for you Freud's views on this phenomenon. Freud describes three layers or stages of jealousy: (1) *Competitive* or normal jealousy; (2) *projected jealousy;* and (3) *delusional* jealousy. Normal jealousy is made up of grief, the pain at the thought of losing the loved object, and of the narcistic wound, one's *amour propre* is hurt at the idea of losing the woman. There is also a feeling of hostility against the successful rival, and a certain amount of self-criticism for losing the object. Although it is called normal jealousy, it is not always entirely rational; *i. e.,* it is not based altogether on actual situations, nor is it proportionate to the real situations or under full control of the ego. It is also to be noted that in many persons it is experienced bisexually. Thus, the jealous man may not only experience pain in regard to the loved woman and hatred towards his male rival, but he may also feel grief in regard to the unconsciously loved man and hatred towards the woman as his rival. Thus, one of my patients, also seen by Professor Freud, suffered from jealousy phantasies, wherein he identified himself with both his successful rival and with his mistress. He went through a regular sort of routine in his phantasies. He visualized the minutest details of coitus between his mistress and her paramour; during the preliminaries he hurled a thousand maledictions on his rival, while during the actual act, he experienced

severe pain at every fancied motion of his rival, and then cursed his mistress for being an insatiable vampire. Such an identification is based on the more complete œdipus complex.[15]

Projection jealousy originates in both man and woman either from their own actual unfaithfulness or from impulses to unfaithfulness, which were repressed. Here Freud indulges in a bit of philosophizing about matrimonial fidelity, which the average man recognizes, but dares not express frankly. He states that the degree of fidelity required in marriage can only be maintained in the face of continual temptations. Anyone who denies this in himself will, nevertheless, feel the impulsion to it so strongly that he will be glad to make use of an unconscious mechanism as an alleviation. This relief, or absolution by his conscience, he attains by projecting his own impulses to disloyalty to his wife, to whom he owes loyalty. Freud adds that social conventions have wisely taken account of this universally existing state of affairs by granting some leeway to the married woman's desire to attract and to the married man's desire to conquer; it is expected that the indisputable tendency to infidelity will thereby be drained and rendered harmless.[16] Conventional experience teaches that such little ventures in the direction of infidelity are not to be held against either party, and in most cases it actually happens that the inflamed passion, fanned by a strange love object, finds gratification in a sort of turning back to fidelity at home. The jealous individual, however, does not recognize this conventional tolerance; he does not believe that one can halt or turn back once one has started on this path, nor does he believe that social flirtation can serve as a safety-valve for real infidelity. Projection jealousy has, to be sure, an almost delusional character, but it is amenable to analysis in that the patient can be made to recognize the unconscious motives of his phantasies. The third form, delusional jealousy, is more severe. It, too, originates on the basis of repressed impulses to infidelity, but the objects of its phantasies belong to the same sex. Delusional jealousy is "an acidulated homosexuality and justly belongs among the classical forms of paranoia." As an attempt at defence against a very strong homoerotic striving (in man) it may be expressed in the formula, "Indeed, I do not love him, she loves him."

In returning to our patient, I cannot do any better than to give you Profesor Freud's summary of the case. He states: "When I saw him, he was still subject to clearly defined attacks which lasted for several days and curiously enough regularly appeared on the day following an act of intercourse, which was incidentally satisfying to both of them." The inference is justified that after every satiation of the heterosexual libido, the homosexual component, likewise stimulated by the act, forced for itself an outlet in the attack of jealousy.[17] The jealousy of the attack was based on his observation of the smallest possible acts, in which the unconscious coquetry of the wife, unnoticed by any other person, had betrayed itself to him. He paid very marked attention to all her acts, she smiled pleasantly or looked attentively at her dinner partner, she lingered too long when the postman delivered the mail, she made frequent visits to the school principal. All these innocent trifles he always interpreted correctly. "His abnormality really reduced itself to this, that he watched his wife's unconscious mind much more closely and then regarded it as far more important than any one else would have thought of doing." Freud then compares this to the behavior of those suffering from persecutory paranoia, who, too, cannot regard anything in others as indifferent, and they, too, absorb the smallest possible indications unintentionally offered to them by strangers into their delusions of reference. The meaning of the delusion of reference is quite clear; the patient expects something like love from every stranger, but he gets nothing of the kind. He is either ignored or he thinks that unfriendly remarks are passed; the preacher's sermon expresses an accusation or an innunendo of some sort. Freud adds: "And when we consider the fundamental kinship of the words, 'stranger' and 'enemy', the paranoiac is not so wrong in regarding this indifference as hate, in comparison with his claim for love."[18] The jealous and persecuted paranoiacs, therefore, project to others what they do not wish to recognize in themselves, but they do not just project, as it were, into the air, where there is nothing of the kind already. They are guided by their knowledge of the unconscious, and displace to the unconscious minds of others the attention which they have withdrawn from their own. Our jealous husband recognized his wife's unfaithfulness instead of his own, and by magnifying it he could keep his own infidelity unconscious. We may also infer

that the hostile persecution which R. perceived from his brother-in-law S. reflected his own hostile impulses against S. Here the most loved of his own sex turned into the persecutor, by means of the ever-present ambivalence of the feelings, which was reenforced by the unfulfillment of his need for love. Ambivalence thus served the same purpose for R., the persecuted paranoiac, as jealousy served for M., our jealous husband; namely, as a defense against homosexuality. M.'s homosexuality showed itself in his whole behavior. He never made any friends or had any interests to speak of. Professor Freud thought that his jealousy delusions constituted his first actual relations with men. He had a weak father and an active mother, a situation which one finds quite frequently in homosexuals.* At an early age, he sustained a homosexual trauma. His shoes were stolen while he was swimming with other boys. The thief, a man of mature age would return them only after the boy submitted to pederasty. This obnoxious and brutal trauma forced his homosexuality into repression and barred the way to its sublimation. He was his mother's favorite and married a rich girl in order to ameliorate her lot. When he became engaged he had first obsessive doubts about his fiancée's virginity (which according to Freud's statement was based on an unconscious longing for a virgin mother) but this soon quieted down and during the first years of marriage he was not disturbed by morbid jealousy. But, very soon after he stopped an illicit sexual affair, which had lasted for about two years, the jealousy delusion appeared and then dominated all his thoughts and actions.

When M. returned to me after his first sojourn in Vienna, I advised him to follow Professor Freud's suggestion and return to him for more treatment. I did not enter into any analysis of his case, but from a dream, which he voluntarily related, I learned that he identified me with his oldest brother, who was a physician and who originally brought him to me. On his second return when Professor Freud asked me to continue the analysis, I soon dis-

*The absence of the father through early death often produces a fixation on the father image, which favors inversion. In such cases the œdipus conflict, which normally establishes the son's behavior towards his sex, is here lacking. In the case of weak fathers of the "hen-pecked husband" type, the same situation results because the son develops a deep longing for a strong aggressive man.

covered that he had always been jealous and afraid of his "doctor-brother." The latter was an aggressive individual, who had displaced the father in the home. I believe that it was this part of the analysis in which he, so to speak, made peace with his brother that gave the finishing touch to the psychosis. I might also add that his brother was quite sick at the time of his analysis with me and died soon after I discharged the patient. A few years later I learned to my surprise that M. was attending the Christian Science Church; I made it my business to meet him "accidentally" and he explained that it was his wife's idea to give the children some sort of religion. He said that he only attended the church very infrequently and made the following remark, "As a man of science, I cannot believe that nonsense." However, I wondered whether Mrs. Eddy represented the last remnants of his sublimated mother complex.

Mention was frequently made of the œdipus complex, with the mechanism of which you are all acquainted; I also spoke of the *more complete œdipus complex,* when I told you about the double identification of the patient who suffered so much in his phantasies of coitus between his mistress and the imagined lover. Now, the simple œdipus complex consists of an ambivalent attitude towards the father and an object relation of a purely affectionate kind towards the mother.[19] When the œdipus complex passes away, the object-cathexis of the mother is given up and the father identification becomes more intense. These changes consolidate the masculine character of the boy and retain a certain amount of affectionate relation to the mother. Briefly, this represents the normal issue of the œdipus situation. If something happens, however, which prevents the boy from fully identifying himself with the father and he instead identifies himself with the mother, his future psychosexual life cannot be normal, and frequently enough he becomes a homosexual. But, whether the outcome of the œdipus situation, in both sexes, is to result in an identification with the father or with the mother depends on the relative force of the masculine and feminine sexual dispositions, in the individual concerned. This is one of the ways in which bisexuality influences the fate of the simple œdipus complex. But, as has already been intimated, there are other and perhaps more significant ways which can only be explained by assuming a more complete œdipus complex. This

form is twofold, positive and negative, and depends on the original bisexuality of the child. The sensitive, neurotic boy has not merely an ambivalent attitude towards his father and an affectionate object-relation towards his mother, but he behaves at the same time like a girl, that is, he displays an affectionate feminine attitude towards his father and a corresponding hostile jealousy towards his mother. In the case of neurotics and psychotics, we are always sure to find the dynamic results of the more complete œdipus complex, which can be readily unravelled in the neurotic, but in the psychotic, who also utilizes dereistic thinking, the situation is often very complicated.

To illustrate the homoerotic processes in some cases of schizophrenic paranoia, I selected the case of G., a young man of 29, who first consulted me during the winter of 1925. He was also seen by Dr. George H. Kirby and Dr. M. S. Gregory and was a few months in the Crichton House under Dr. Lamb's care. For the last two years he has been a patient in a U. S. Veterans' Hospital for Mental Diseases. Mr. G. was the second of four children; he came between two brothers and the fourth child was a girl. Without going into a detailed history I will state that the patient was paranoid for years before we saw him. His main persecutory delusions revolved around his father, who according to the patient had him watched by detectives. Wherever he turned, he saw his father's detectives. The patient gave the following reason for the persecution: His father had homosexual feelings for him. His father never lived happily with his mother so he centered his affection on him. The patient frankly admitted that he, himself, had homoerotic feelings and experiences; still, he was not sure whether he should accede to his father's homosexual wishes. His father never spoke directly of his homosexual desires, but he knew that his father wanted him to take a trip with him in order to find out whether he was willing to enter with him into homosexual relations. Realizing that I might not believe his detective stories he showed me an affidavit signed by a lady friend, D., before the American Consul in Paris, stating that frequently while in his company in Paris she saw detectives following him everywhere. Mrs. D. corroborated to me her former statement and added that the same thing also happened here in New York. She insisted that she repeatedly saw detectives following G. and her. We were

obviously confronted with a case of *Folie à deux*. Mrs. D. was about 10 years older than the patient; she belonged to the emancipated type of women and was just getting a divorce from her husband with whom she had not lived for many years. She apparently was in love with the patient.

The patient showed a tendency to systematize his delusions. His father was very active in the Episcopal church, but he was also mixed up with a secret phallic cult. In order to seduce him to homosexuality, his father kept on sending homosexuals, who made advances to him, and he invariably interfered and somehow broke up any normal relations with women that the patient attempted. He readily admitted, too, that he had homosexual experiences with his father's "emissaries" and that he was "more or less" bisexual from early boyhood. As a result of being harassed by the father, he was forced to send him telegraphic messages, in which he threatened to take decisive measures against this persecution. Meanwhile, the patient corresponded, and I might say pleasantly, with his father, to whom he was beholden for very generous monthly allowances. The latter, too, was elaborated in his delusions, as his allowance he usually spent within a few days and then went into debt.

Concerning the patient's past history, we can state that it may be characterized by a marked emotional instability. As a child, he was delicate and ailing, so that he received more than his share of love from his very neurotic mother, but his physical development was normal. His mother stated that from early childhood he had always been very wilful and subject to tantrums of irritability and crying, so that he did not get along well with the other children. He was always considered bright and intelligent, but was a poor student. He did not like anything that required persistent effort, but he showed considerable talent in drawing and as he grew older, he wished to be an artist. His whole development and attitude and manner towards life reminded me of the typical invert, or as Ferenczi would call him, the subject homoerotic. This was undoubtedly also favored by the fact that the mother was very desirous of having a girl when he came as the second boy. His mother also stated that as a boy, he was very sensitive and very jealous of his father and older brother, and as he grew older, he became more and more secretive. He had a habit of hiding whatever was

his and was suspicious of everybody. Sexually he was always very inquisitive and his mother recalled many episodes to substantiate this. She herself, was a very prudish woman and tried in every way to curb this sexual curiosity through all kinds of punishment. His father was a very busy person, a successful man, and paid little attention to the patient until he was about 10 years old, but from this age until the patient was 14 there was a very close relationship between them. But, at this age his father insisted that he go to a prep-school far away from home, for which the patient never forgave him. He went through school with much difficulty. He hated his studies, wanted to be an artist, and showed good talent in drawing and painting. Following his graduation from school, he held a few jobs for short periods and then entered an art school in Chicago where he was on and off for about two years. When the World War came, he enlisted in a French aviation unit, and later transferred to the United States Aviation Corps, where he remained until the armistice was signed. His services were uneventful. When he returned home, he showed a marked restlessness and soon decided to go back to Paris. There, he led the Bohemian life of the left bank of the Seine; he was supported by his father and he was supposed to study art. Actually, he just idled away his time in cafés and restaurants. In 1931 he returned to his home and showed the paranoid system complex, which finally resulted in his commitment. In the Crichton House he was well behaved, but could not hide his delusions, and as his parents wanted him to get another chance in the outer world, he was allowed to leave the sanitarium after about two months. He quickly married Mrs. D., who by this time was fully apprised of his insanity and of his abnormal sexual life. She, however, refused to be influenced by my admonition; she said she was not jealous, she loved him, and wished to help him.

All the psychiatrists, who saw him, diagnosed him as a paranoid præcox. In contradistinction to the cases cited above, our patient's sexual life remained on an infantile level. I have no time to go into a detailed description of it, but I can state that to all outward appearances, he was bisexual. He, himself, thought he was about 30 per cent homosexual, and 70 per cent heterosexual, but these "extremes," as he designated them, seldom gave him any satisfaction, so that sexual indulgence was not a spontaneous activity with

him. He was sought by object-homoerotics which accounted for his more frequent homosexual indulgences; and now and then, an older aggressive woman or a very slender, boyish type caught his fancy, and then he had heterosexual relations. Mrs. D. belonged to the latter class, she was 10 years older than the patient and showed masculine secondary sex characteristics.

From what I could discover about his psychosexual evolution, this patient like all schizophrenics, never detached himself from his infantile polymorphous perversity. His ego was either constitutionally or evolutionally much below par, so that the œdipus situation never actually became dissolved. On the surface he was bisexual; that is, he could identify himself with both parents, but on at least a few occasions he had himself flagellated, and now and then, he resorted also to other algolagnic activities with both men and women. But, in all these activities one could discern a definite weakness; there was nothing spontaneous or clean-cut in his sexual behavior. Very soon after he married D., he developed delusions in which he involved her with his father. He (the father) tried "the same old tricks," he wanted to alienate her from him. Analysis showed that his wife was identified with his mother, his sister, his sister-in-law, and with two women he formerly wished to marry. All of them had been under the domination of his father. One of these girls "died mysteriously" as a result of his father's machinations because "she knew too much." This antagonistic behavior of his father was easily traced to his early jealousy of his father and older brother. The latter, too, was his childhood rival. He fought him almost daily until the age of five. This identification continued because his brother was in his father's business; he was his "right hand man." Whenever G. had occasion to refer to his brother, he always added, "Oh, he does what he is told!" In rational homoeroticism such an intense older brother rivalry frequently plays a leading part in the development of the inversion. For, it is well known that early childhood rivalry derived from the mother complex may be later repressed as a result of training, and the cognition that one can obtain more outlet through loving than hating. The erstwhile rival may then become the first homosexual love-object. As mentioned above, G. first hated both his father and his brother, and later became reconciled to them. His close relation to his father was especially

noticeable. The hated rival apparently became the first love object. But, later when this had to be repressed and projected outward, the wish became changed into: "No, I do not love him—he loves me homosexually, but I do not wish to yield to him, hence he persecutes me." We have already said that this patient was fixated on his mother and identified with her; not only did he resemble her physically, but he had her temperament and, like her, he was artistically endowed. Analysis showed that the patient's protest against serving as a love-object for his father concealed the infantile wish to take his mother's place with him or to share it with her.

Many episodes could be mentioned to show the coöperation of bisexuality (patient's effeminacy) and the more complete œdipus complex in this double identification, but the *dénouement* of his marital life will suffice as the best illustration. A few months after the marriage, his wife called on me and begged me to assist her to get away from G. She was fully convinced that he was insane and as a corroboration of it, she related the following strange episode: About a month before, G. brought home a former buddy, whom he met accidentally in a speak-easy. As a result of considerable alcoholic indulgence, she finally acceded to her husband's request that they all go to bed together. This situation of *à trois* was repeated on a number of occasions for some weeks.

The patient then frankly told me all about it. In fact, he wrote out everything under the title of "Summary of mixed and diversified sexual experiences." The man whom he called John had had homosexual relations with him during the war; he was his commanding officer. Hence, his father substitute, and the wife was a typical mother substitute. Following the more complete œdipus complex, to which we must here add the factor of bisexuality, G. lived through in these acts both the identifications; he was the father and the mother. The patient's description of this strange sexual episode plainly shows the wish realization of the more complete œdipus complex. He writes: "The best party I have ever had, without exception. But, here is the most important element about the situation. I was more or less in control of things. It was *my* woman and I had a greater factor of masculinity on my part and I loved them both. She was not trying to get him as a permanent proposition, nor he her. She was clever in making me feel by her action as follows: 'You (myself) are the one I really am

attached to; I love your friend as long as you love him, my universe is your universe and what you want shall happen.'" Delicacy prevents me from quoting all the details as given by the patient, but any one reading them can plainly see the unconscious mechanisms of the patient's delusions. He sought in every way to defend himself against his passive homosexual temptations which corresponded to his early attitude to the father by reacting to them with the delusions of persecution. At the age of six, he slept with his father on a few occasions and through the latter's innocent but rather loving behavior the patient sustained a homosexual trauma which fell on fertile soil and undoubtedly revived his early infantile cravings for the father. Another fragment of the above description states that he also acted passively towards John during the "mixed and diversified sexual experience." Such a triple relation is not unknown in sex pathology. The French refer to it as *ménage à trois,* and the sexologists designate it as *troilism.* Magnus Hirschfeld states that troilism is a disguised homosexuality.[20] Nor is troilism altogether foreign to everyday life. This year's most successful play in New York City revolves altogether around this subject. The *dramatis personæ* consist of two men and a woman. The men love each other and both love the woman. She lives alternately now with one and now with the other. A third man, an acquaintance of all, comes along and induces her to marry him. After living with him for a short time the two erstwhile lovers make their unexpected appearance, and she readily consents to leave her husband and go to live with them both. When her husband protests, she expresses herself in some such formula as this: "I love him (pointing to one of the friends) and I love him (pointing to the other) and he loves him (pointing from one to the other), and they love me." And, she forthwith leaves with them. I do not know whether the author of the play knows our psychological view of homoerotism, but all the essential factors of homoerotism are cleverly depicted. The narcissism was expressed by making the two men look and dress almost exactly alike; they use the same words and expressions in an echolalic manner, and they even kiss each other. The over-valuation of the penis, which is one of the essentials of homoerotism, the play expresses in the following witticism: The hero and heroine are discussing the criticism of the hero's play in the London *Times.* The heroine does

not think that it matters much, to which the hero says " But, don't forget, it is in the London *Times*— it's the *organ* of the nation." (Explosive laughter from the audience.)

For obvious reasons no attempt was made here to give the deeper mechanisms that enter into the analyses of such cases. Those who are interested should refer to the authors mentioned and to the works of Abraham, Bibring, Ruth Brunswick, Fenichel, Jones, Nunberg, Stärke, Van Ophuijsen, and others. Nor, was it possible to give here numerous cases to show the various gradation from normal to pathological homoerotism. But, it is quite plain that the adjustment of modern men is essentially homosexual. Whether it concerns the struggle for bread, or for women, men are forever rivals. The task of civilization, as represented through all religions, has always been to do away with rivalry and hatred. Christianity stands in the foreground in this respect. We are admonished to love our neighbors as ourselves, and even to love our enemies. In the light of everyday occurrence, who dares say that civilization has been successful in accomplishing its task. As long as the individual does not suffer from the stress of need, he can remain calm, but as soon as economic or sexual struggles reach a certain intensity, all precious rules are thrown aside. In the beginning of time men warred for hunger and sex. The ancient Greeks fought over Helen of Troy, and Genghis Khan's hordes over-ran the civilized world in search of bread. Historians tell us that the prettiest women failed to move these warriors. But the dawn of civilization began when the Sabine ladies hurled themselves between Roman and Sabine armies, and by proffering love to both, stopped the armies from killing each other. The erotic or life instinct thus replaced the destructive or death instinct. For it is the former that unites and promotes love, and in sublimated form, all the great assets of culture. Pathological homosexuality, while permitting much play to Eros, curtails its activities to a fatal extent. It is a narcistic outlet at best. Paranoia is a first cousin of homosexuality in that it, too, is an offshoot of narcism, but its regression to the anal-sadistic level (Stärke and Van Ophuijsen) enables the destructive impulses to push the erotic instinct to the background and thus make way for the sadism of the death instinct.[21]

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A TUBERCULOSIS SURVEY IN MENTAL HOSPITALS.*

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Physicians in charge of institutions for the care of mental cases have learned that tuberculosis may assume formidable aspects among patients, and may become a serious menace to the staff due to the intimate contact of such patients with nurses and attendants.

It has been generally thought and often stated that a relatively high proportion of mental patients suffer from pulmonary tuberculosis. The mortality due to tuberculosis in some mental hospitals has far exceeded that in the general population.

TABLE 1.

Comparison of Percentages of Deaths Due to Tuberculosis in Relation to Deaths from all Causes Among Patients in Mental Hospitals and in the General Population of Ontario.

Year.	Total deaths.	Tbc. deaths.	% deaths due to tbc.	% of tbc. deaths to all deaths in general population.
1928.....	690	60	8.69	4.94
1929.....	741	79	10.66	4.46
1930.....	725	66	9.10	4.89
1931.....	655	39	5.95	4.61
1932.....	813	68	8.36	4.57

In order to secure some definite information as to the incidence of tuberculosis, especially that of the pulmonary type, in the Ontario Mental Hospitals, arrangements were made in November, 1930, to conduct a survey of selected cases. The patients were chosen by the staffs of the hospitals and included:

(a) Those known to have tuberculosis.

(b) Those suspected of having tuberculosis.

(c) Those with history of known contact with tuberculosis, either prior to or after admission to hospital.

* Read at the eighty-ninth annual meeting of The American Psychiatric Association, Boston, Mass., May 29–June 2, 1933.

† By invitation.

Eight hundred and eighty-six (886) adult patients were selected for examination out of patient population of 10,500. An X-ray film of the lungs was taken on all cases. Approximately two-thirds of the total group were given a physical examination and kept under observation for a given period.

Six hundred and ninety-two, or 72.4 per cent of patients, showed X-ray shadows within the usual limits of normal, absence of pyrexia and symptoms and were classified as "Tuberculosis not thought to be a factor."

Table 2 is a summary of our findings.

TABLE 2.

CLASSIFICATION OF THE TUBERCULOUS CASES FOUND IN 886 SELECTED ADULT PATIENTS IN HOSPITAL POPULATION OF 10,500 (10 HOSPITALS).

	Minimal arrested not requiring isolation.	Requiring isolation.		
		Minimal.	Moderately advanced.	Advanced.
Males	14	17	26	13
Females	43	22	31	13
Total	57	39	57	26

Total requiring isolation 122, or 13.7 per cent of patients examined.

Seventy-one (71) females and 51 males, or 13.7 per cent, showed slightly increased X-ray shadows, pyrexia and unexplained symptoms. These were classified as requiring further observation, which was to include a repeat X-ray of the lungs and physical examination within one year.

The Orillia Hospital is for the care of mental defectives and at the time of the survey had a population of some 1400 patients. Four hundred and thirty-two of the patients were under 17 years of age. All of these children were tuberculin tested by the Mantoux or intracutaneous method. This diagnostic procedure is of value in determining the presence or absence of infection with the tubercle bacillus.

A positive tuberculin reaction indicates that infection has taken place but does not indicate that the infection has progressed to the stage of tuberculous disease. Further, a negative tuberculin reaction, provided sufficient dosage is given, may be interpreted as

indicating the absence of infection or tuberculous disease if we except far advanced active cases nearing fatal termination.

Two hundred and fifty-six, or 59.2 per cent, of the total group of 432 boys and girls gave positive reactions to tuberculin.

These figures are apparently higher than those obtained in a survey of rural and urban school children of similar age groups carried out in Ontario, at which time 34.8 per cent of the children tuberculin tested gave positive reactions. The possible presence of tuberculous infected cattle in the hospital herd several years ago may have adversely affected the percentage of reactors among the mentally defective children. Bovine infection will give a reaction to tuberculin produced from the human strain.

The 256 positive reactors were X-rayed along with 100 other selected cases over 16 years of age.

Table 3 gives the classification of the tuberculous cases found.

TABLE 3.

CLASSIFICATION OF TUBERCULOUS CASES FOUND IN ORILLIA HOSPITAL, 1931.

	Hilum.	Minimal.	Moderately advanced.	Total.
Girls under 16.....	1	0	0	9
Boys under 16.....	4	3	1	
Adult female	1	5	3	12
Adult male	0	1	2	
				21

All cases considered active.

Two wards were set aside in the Orillia Hospital for the revealed tuberculous mental defective cases. In the following two years only two new cases of tuberculosis have been discovered in this hospital. It is worth noting here that 150 of the negative reactors in 1931 were re-tuberculin tested this spring and 13 per cent of this group reacted positively. This finding indicates that in some manner these patients have been in contact with tuberculosis in the interval. However, comparing this group of negative reactors in 1931 and some two hundred new admissions in the same age groups, it was found that a higher percentage of reactors was obtained among the new admissions. This would lead us to believe that the possibility of infection in an institution where measures for the prevention of infection exist, even if not perfect, is less than among

the general population. This is in accordance with the finding of A. N. Bronfenbrenner, "Tuberculous Infection in Mental Defectives," *Amer. Rev. of Tbc.*, March, 1932.

Survey of Staff.

All the nurses in the 11 provincial hospitals and 39 selected members of male staff were X-rayed and about one-half were given a physical examination.

The findings are shown in Table 4.

TABLE 4.

	Total X-rayed.	No. receiving physical examination.	No. requiring further observation.	No. found to have tbc. disease.
Nurses	798	452	30	12 or 1.5% 8 had active disease
Male staff	39	34	3	5 or 8.6% 3 had active disease

The three active cases in the male staff were physicians.

It was felt the percentage of nurses found to have active tuberculosis at the time of the survey did not give a true picture of the incidence of disease in this young female adult group. Just previous to the survey five nurses had been sent to sanatoria and in the following nine months eight more nurses were found suffering from tuberculosis.

At the completion of this survey one entire building recently renovated with accommodation for 125 patients was set aside in the most centrally located hospital in the Province (Mimico) for the treatment of the adult tuberculous cases from all other hospitals. During the following year each hospital reported the finding of a number of additional tuberculous cases requiring isolation and treatment. These patients had not been included in the previously examined group.

It was the opinion of those interested in the work that possibly many more tuberculous cases had been overlooked and because of this the most representative of the provincial hospitals (London) was chosen for the purpose of carrying out complete examinations of all patients and staff.

The patient population of the London Hospital was 1209 and the staff numbered 278. It was hoped that by such a survey a

reasonably accurate picture could be obtained of the probable incidence of tuberculous disease among the staff and patients of all mental hospitals.

With the view of lessening the expense of X-ray pictures and the saving of time, all patients and all staff were tuberculin tested. The tuberculin used was prepared by the Connaught Laboratory of the University of Toronto and standardized against the international standard as supplied by the committee of standards of the Health Committee of the League of Nations. The intracutaneous method was employed. As indicated in Table 5, approximately 90 per cent of patients showed evidence of infection.

TABLE 5.

TUBERCULIN TESTS—PATIENTS.

1/10 c. c. containing 1/10 mg. of tuberculin.

Total number in group	1209	
Positive reactors	1107	or 91.5%
Negative reactors	102	or 8.5%

CORRELATION WITH SEX IN PATIENTS.

MALE.

Total number in group	567	
Positive reactors	515	or 90.8%
Negative reactors	52	or 9.2%

FEMALE.

Total number in group	642	
Positive reactors	592	or 92.2%
Negative reactors	50	or 7.8%

Each of the eleven hundred and seven (1107) patients reacting positively to tuberculin received an X-ray of the lungs. Those showing shadows indicating definite tuberculous disease and those with shadows beyond the limits of normal, even if the shadowing suggested non-tuberculous conditions, were given a physical examination and kept under observation for a period of two months during which time temperature records were kept.

Table 6 is a summary of the findings in this group.

For the protection of nurses and attendants 15 of the apparently arrested cases were classified as requiring isolation because of uncontrolled cough and expectoration even when repeated sputum examinations failed to reveal tubercle bacilli. It was thought that these patients were potential sources of infection to contacts.

Because of X-ray findings 56 other cases were classified as "requiring observation" which was to include periodic temperature records and repeated X-ray in three, six or 12 months' time.

From the above table it is reasonable to conclude that tuberculous disease is more prevalent among patients in mental hospitals than

TABLE 6.

CLASSIFICATION OF TUBERCULOUS CASES FOUND AMONG 1209 PATIENTS (LONDON).

	Number.	Active.	Apparently arrested.
Minimal	63	26	37
Moderately advanced	18	16	2
Advanced	3	3	0
Mediastinal or hilum	4	0	4
	88 or 7.3%	45 or 3.7%	43 or 3.5%

All the active cases (45) and 15 of the 43 apparently arrested cases required isolation.

Cases requiring isolation—60, or 4.95 per cent of patient population.

Cases in which isolation not thought necessary—28.

in the general population or in patients treated in general hospitals. This might be attributed to several factors:

1. An open case of tuberculosis with uncontrolled cough and expectoration is a potential source of infection to all other patients on the ward. This group is likely to be materially larger than the number of contacts found in the homes of the same tuberculous. Overcrowding of patients and poor ventilation accentuate this factor.

2. In a large proportion of mental patients the circulation is congested and metabolism at a low ebb. A generally under par physical condition is often present with lowered resistance. In the 88 tuberculous cases found the hearts were small from slight to marked degree in 20 cases or 22.7 per cent.

3. The difficulty experienced with some patients to insure the intake of food in proper quantities.

Physicians, nurses, female and male attendants, farm help and clerical staff numbered 278.

Each member of staff was tuberculin tested. Tables 7 and 8 illustrate the results of the tests.

TABLE 7.

TUBERCULIN TESTS—STAFF.

MALE AND FEMALE.

Total number in group	278
Positive reactors	208 or 74.8%
Negative reactors	70 or 25.2%

FEMALE.

Total number in group	147
Positive reactors	94 or 63.9%
Negative reactors	53 or 36.1%

MALE.

Total number in group	131
Positive reactors	114 or 87.0%
Negative reactors	17 or 13.0%

The higher percentage of positive reactors among the male group is due to the small number under 25 years of age.

TABLE 8.

TUBERCULIN TESTS—NURSES "FEMALE."

CORRELATION WITH AGE.

		Positive.		Negative and doubtful.	
Age.	Number.	Number	%	Number.	%
15-19	38	22	58	16	42
20-24	56	29	52	27	48
25-29	19	18	94	1	6
30-34	6	3	50	3	50
35-39	6	6	100	0	0
40-44	6	4	67	2	33
45-49	3	2	66	1	34
50-54	6	5	83	1	17
55-59	3	1	33	2	67
60-plus	4	4	100	0	0

This table brings out two points that are extremely interesting, namely: (1) Approximately 50 per cent of young women entering the nursing profession have not been infected with tubercle bacilli and, therefore, have no specific immunity against infection;

(2) The high percentage of nurses who apparently received the infection between the ages of 25 and 29. These nurses receive their original infection after beginning their training and most probably within the hospitals.

These out of the ordinary findings were confirmed by extending the tuberculin testing to include all the nurses associated with the various provincial hospitals, 794 in all, of whom 104 were under 20 years of age. Of these only 47.1 per cent gave positive reactions to tuberculin.

After the use of tuberculin in varying amounts and dilutions it was determined that 1/100 mg. of tuberculin in 1/10 c. c. solution was the preferred dose in initial tests. We also found that the negative reactors could be further tested with amounts up to 1 mg. in 1/10 c. c. without danger of serious reactions. If negative to 1 mg. one can definitely rule out the possibility of infection being present. The reactions should be read 40 to 48 hours after the injections.

X-rays films of the lungs were taken on the 208 members of the staff giving positive tuberculin reactions and of these 75 received a physical examination. The results are shown in Table 9.

TABLE 9.

CLASSIFICATION OF TUBERCULOUS CASES FOUND IN 278 STAFF (LONDON).

	Mediastinum.	Minimal.	Moderately advanced.
Nurses, 147	1 arrested	1 active	1 arrested
Male, 131	0	1 active	1 arrested

The three tuberculous cases among the females were nurses and the two among the males were attendants. The two nurses with arrested disease had been discovered during the previous survey and had been permitted to return to duty. Five other nurses and one physician are now under observation because of suspicious signs and symptoms.

Before preventive measures were instituted in the various hospitals, members of the staff were frequently discovered with moderately advanced disease.

Since segregation of the tuberculous patients has been brought about and more careful examination, including X-ray of the lungs, of nurses entering training, fewer patients and nurses have been discovered developing tuberculosis and these in the minimal stage with every expectation of making a complete recovery.

It has, therefore, been made an administrative policy to examine (including X-ray) all new employees and nurses entering training as well as patients showing any suggestive signs.

Following is the mental classification of the tuberculous patients:

TABLE 10.

MENTAL CLASSIFICATION OF TUBERCULOUS CASES IN ONTARIO HOSPITALS.

Mental diagnosis.	Tbc. cases.	% of those found tbc.	% of various psychoses, first admissions, over the past five years.
Dementia præcox	89	47.83	27.6
Paranoid states	36	19.35	2.3
Manic depressive	19	10.2	12.5
Mental deficiency	13	6.98	4.1
General paresis	8	4.30	5.8
Toxic psychoses	4	2.15	.51
Senile psychoses	4	2.15	11.5
Epilepsy	3	1.61	2.7
Involution melancholia	2	1.07	4.3
Exhaustion psychoses	1	.53	.9
Psychoneurotic	1	.53	1.4
Undiagnosed	6	3.22	1.2
	186		

It will be noted from Table 10 that the percentage of tuberculous patients found among the præcox and paranoid groups was excessively high. One would rather expect this finding in the chronic præcox group. The same finding in the paranoid group might indicate that the condition was due to prolonged hospitalization in an environment from which open cases of tuberculosis had not been previously removed, rather than because of the inertia and low resistance of these particular groups.

Conclusions.

1. A higher percentage of tuberculous disease is found in patients with mental conditions than in the patient population in general hospitals or in the general population exclusive of certain industrial groups (miners in quartz rock, stone cutters, etc.).

2. Infection with tubercle bacilli has taken place in a higher percentage of mental defective children than in apparently healthy school children.

3. Because the constitutional symptoms in certain mental conditions are similar to those found associated with tuberculosis, every effort should be made to rule out tuberculosis as the cause of the symptoms in all suspicious cases.

4. Temperature records are of value but other possible causes of pyrexia should be eliminated. Most excited and some depressed mental cases have pyrexia of unexplained origin from time to time.

5. Repeated sputum examinations on all suspicious cases should be carried out whenever possible. It is very difficult to obtain sputum for examination from a large proportion of mental cases.

6. Weight charts should be kept on all tuberculous cases and those under observation.

7. All active tuberculous cases and a large proportion of the arrested cases in mental hospitals should be isolated.

8. The apathy and indifference to physical discomfort is found to such an extent in a large percentage of mental cases that cough and expectoration are frequently absent and loss of weight and strength with pyrexia are often the first sign indicating the advisability of chest investigation.

9. Because of poor cooperation in physical examination on the part of a large proportion of mental patients, an X-ray of the lungs is essential before the probability of tuberculosis can be eliminated. All cases suspicious of having tuberculosis should be X-rayed.

10. An intracutaneous tuberculin test properly done is of value in determining the presence of infection especially in the younger age groups. We have previously stated that no reaction to 1 mg. of tuberculin in 1/10 c. c. definitely rules out the possibility of infection. However, for all practical purposes a negative reaction to 1/10 mg. in 1/10 c. c. solution may be accepted as indicating the

absence of infection or disease if we except the almost moribund tuberculous cases.

11. X-ray pictures of the lungs should be taken on all probationer nurses before they are accepted.

12. Morbidity from tuberculosis is high in the nursing profession.

13. A much smaller percentage of young women under 25 years of age have been infected with tubercle bacilli than was formerly believed. These nurses with no pre-existing infection and resulting immunity should not be given duty on wards with known open cases of tuberculosis.

Our thanks are due to the superintendents and staff of the Ontario hospitals for their cooperation and assistance in completing the survey.

THE BLOOD SUGAR IN RELATION TO EMOTIONAL REACTIONS.*

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Many articles have been published concerning the blood sugar in psychotic states. There has been a widespread expectation that emotional reactions should be characterized by hyperglycemia, as in Cannon's classical experiments [1] on laboratory animals. The conflicting evidences and opinions concerning emotional hyperglycemia were adequately presented by Bowman and Kasanin in the communication [2] setting forth their own observations, which were essentially negative.

I wish to report herein the findings on a considerably larger group of mental patients (958) than any hitherto reported, and to discuss the physiological significance of the observations. The material presented in this communication has been collected during about 12 years of study of emotional reactions in the patients of a relatively small mental hospital. The figures here given are all fasting blood sugar concentrations, that is, they represent the copper-reducing [3] capacity of venous blood, taken in the morning approximately 13 or 14 hours after the last preceding meal. The analytical technique has been, for the larger number, a uniform modification of the method of Folin and Wu. In the last few years, the 1930 copper method of Folin for unlaked blood [4] has been regularly used, which yields figures appreciably nearer the true glucose concentration, averaging in our experience about 15 mg. per cent lower than those by the Folin-Wu method. For my own satisfaction I have considered the two sets of figures separately, but finding no significantly different conclusions, I have combined all into a homogeneous set of data by arbitrarily adding 15 mg. per cent to all sugar determinations on unlaked blood.

In the early years of this study, beginning in 1921, careful attempts were made to secure from physicians and nurses an ade-

*Read at the eighty-ninth annual meeting of The American Psychiatric Association, Boston, Mass., May 29–June 2, 1933.

quate account of the patients' affective reactions, in the hope of establishing some system of gradation for correlation with the blood sugar figures. This naïve hope has not been fulfilled. At times, when a few dozen figures had accumulated, from which I could extract no very definite meaning, I also used to submit them separately to different members of the staff more familiar than I with the patients. Reasons could commonly be found for believing that those patients showing the somewhat higher blood sugar figures were under genuine emotional tension; but if the blood sugar figures were not revealed, about equally good reasons could be adduced indicative of genuine emotion in the others. When fictitious figures were submitted, fairly good correlation could then also be made out. This is not said in criticism of those who so kindly helped me; but rather to illustrate the unreliability of judgment or consensus of opinion regarding the depth or genuineness of minor emotional reactions. To avoid these uncertainties it has seemed wiser to consider as of critical importance for the theory of emotional hyperglycemia only those very extreme reactions concerning which there could be no doubt.

Observations on Very Excited Patients.

It has been our good fortune to get 32 fasting blood sugar determinations on occasions of extreme excitement. I do not mean by this merely the talkative and overactive manic state, but rage-like reactions of the extreme type exemplified in incidents like these:

1. A young schizophrenic man, ordinarily quiet though sometimes sarcastic, was denied breakfast. He cursed and shouted at the nurses, attacked them with a chair, which he smashed to pieces. A blood specimen, obtained a few minutes after this fit of rage began, contained 103 mg. per cent sugar. (About a month before, while quietly cooperative, his fasting blood sugar was 116 mg. per cent.)

2. A young manic woman, ordinarily talkative and much confused, was found, at the time for taking the fasting blood, crouched in a corner of her room, yelling and spitting at the nurses. Her limbs were trembling, her face flushed and her pupils widely dilated. She struggled violently with the nurses for a few minutes to escape some vaguely-expressed danger, apparently, in her mind, a sexual assault. Blood sugar 90 mg. per cent.

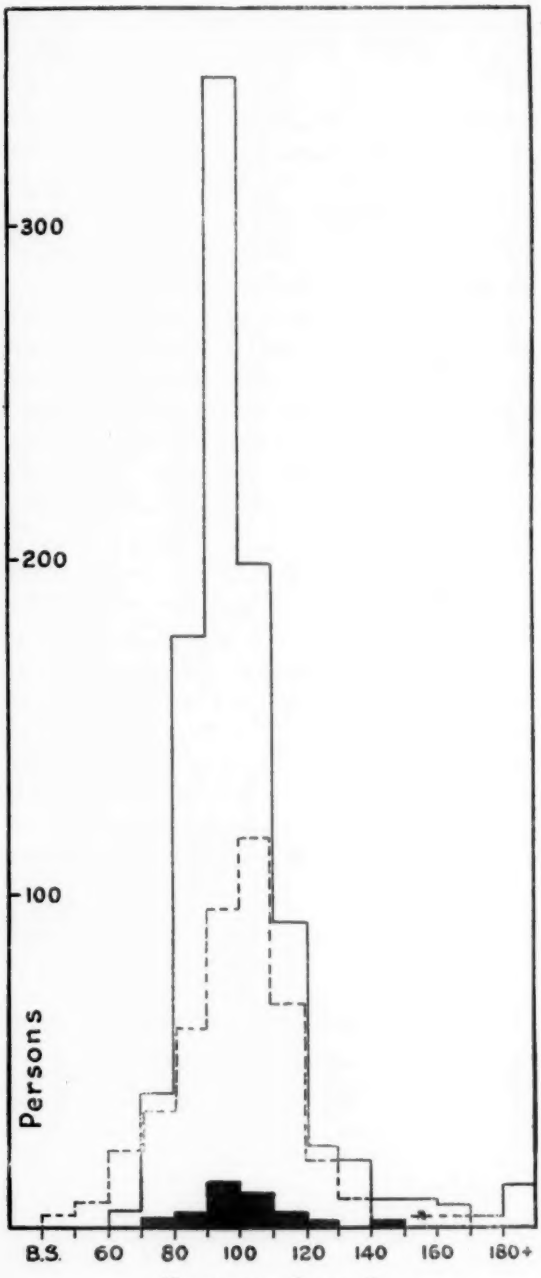
3. A powerfully-built manic man of 44 years, naked, shouting obscene taunts and threats. He struggled violently with five nurses and expressed regret that he did not succeed in maiming any. Blood sugar 108 mg. per cent. (His fasting blood sugar, on quiet occasions and in the normal state, ranged from

111 to 125 mg. per cent. Once, when he was much excited, but not to the extreme degree which constituted our criterion of "very excited," he had a blood sugar of 95.)

These examples indicate the kind of incident listed in this paper as "very excited." I have figures for the fasting blood sugar on 32 such occasions, in 29 different subjects. One showed a blood sugar of 148 mg. per cent (see Case 1) one was 120 and the other 30 were between 75 and 118. In Chart I, the distribution of these 32 figures is compared (1) with the "normal" blood sugar figures collected by Gray [5] and (2) with the results on the total of over 900 persons studied here (excluding 13 unquestionable diabetics, but including a few who could probably be classified as mild diabetes). This comparison obviously gives no support to the hypothesis of an emotional hyperglycemia in excited mental patients comparable to the hyperglycemia produced, in laboratory animals through the adrenal mechanism, when extreme emotional reactions are aroused.[1]

It is not possible to state with complete finality that there is no such a phenomenon as emotional hyperglycemia in psychotic patients. Perhaps it did happen in the case of the one woman whose fasting blood sugar was 148 mg. per cent on one of the two occasions when she was "very excited" at the time of the venipuncture. Perhaps, also, in another thousand patients one might find another such case. But my experience impels me to state emphatically that hyperglycemia is not one of the regular physiological features of extreme emotional excitement in psychotic states.

I cannot, in strict logic, extend this statement to cover extreme emotional excitement in non-psychotic persons, for they may conceivably resemble cats more than psychotic patients in this respect. But I feel justified in expressing a considerable degree of skepticism. I have had one opportunity for such study. One of my children, a healthy boy of four years, having accidentally wounded his foot, received prophylactic tetanus-antitoxin, which resulted in marked urticaria. When preparations were made for the injection of epinephrin he became very much frightened, cried and struggled. His reaction was intentionally aggravated to find out if his adrenal glands could be stimulated to produce enough epinephrin to relieve the urticaria. Although he became very excited, no such effect could be observed. His blood sugar (non-fasting capillary blood)



FREQUENCY CHART I.

——— Psychotic. ■ Very Excited.
------ Gray's Clinical Normals.
(See Tables 1 and 2.)

determined by an improvised micromethod, appeared to be 92 mg. per cent. (An intramuscular injection of 0.15 cc. of adrenalin chloride, 1 : 1000, promptly relieved his urticaria.)

Observations on the Whole Group of Patients.

When one turns from the consideration of the data on "very excited" patients, and examines the collected figures on all patients studied, as presented (with the exception of 13 diabetics) in the frequency distribution in Chart I, the most striking feature is the steepness of the distribution curve, that is, the statistical tendency for the figures to be near 95 mg. per cent. When one considers that many of these persons were exhibiting moderate degrees of emotional disturbance it is again evident that hyperglycemia has little, if any, connection with emotion. Indeed it would appear, on the surface, that psychotic subjects showed more uniformity within the normal blood sugar range than do the "clinically normal" subjects of Gray's collection. I am inclined to discount the unusualness of this uniformity, however, and to attribute it rather to the uniformity of analytical technique used in our series.

Of the 42 persons who showed at any examination a fasting blood sugar concentration of 140 mg. per cent or greater, 13 can be classified beyond reasonable question as diabetic, having shown this disorder of metabolism, in readily demonstrable degree, long before the onset of the psychosis or in later non-psychotic periods, or over such a long period of ordinary emotional condition as to preclude any emotionalistic interpretation of the hyperglycemia. Several others who showed a fairly persistent hyperglycemic tendency are most probably mild diabetics, but they are not so classified here because it seemed fairer for our purpose to err, where errors may occur, in the direction most favorable to the hypothesis of emotional hyperglycemia.

In studying the blood sugar values on these 945 persons, who are "non-diabetic," I have constructed a number of statistical tables, of which only the more significant are here presented, to elucidate the relationship of the fasting blood sugar to age, sex and psychiatric condition. Wherever a number of blood sugar figures on the same person are available, I have combined them into one representative figure. Where all values on the same person are below 140, I have

taken the arithmetical average; where any subject had on any examination a blood sugar above 140 the higher figures are taken as the representative value, because the subsequent lower figures on the same person were often the result of specific treatment, either by diet or insulin. (There are six of our patients who have had to be counted in both manic-depressive depressed and manic-depressive manic groups, so that the totals in the tables of non-diabetics are 951 rather than 945.)

Age and Sex.

From the point of view of that small minority who showed hyperglycemia, the most striking correlations are with age and sex. (See Tables 1 and 2.) Among 484 patients aged 45 years or more, there

TABLE 1.

Frequency Distribution of Fasting Blood Sugars in 951 Psychotic Patients.

(Excluding 13 Unquestionably Diabetic, But Including 6 Duplications of Manic-Depressive Patients as Manic and Depressed.)

Blood sugar mg. per cent. / Age.	40-49	50-59	60-69	70-79	80-89	90-99	100-109	110-119	120-129	130-139	140-149	150-159	160-169	170-179	180 and over.	Totals.
10-19..	..	..	1	4	10	14	11	1	..	..	..	..	..	..	..	41
20-29..	..	..	2	14	39	66	26	10	3	3	1	..	..	..	..	164
30-39..	..	..	..	5	48	72	41	16	5	..	..	..	..	..	1	188
40-49..	..	..	..	4	30	68	35	13	6	6	1	..	1	..	..	164
50-59..	..	..	1	6	23	66	49	27	1	5	1	3	3	1	4	190
60-69..	..	..	..	3	20	35	33	18	5	3	3	2	1	..	3	126
70-79..	..	..	..	3	7	20	18	2	3	2	..	..	..	..	2	57
80-89..	..	..	..	1	1	7	6	4	..	..	..	1	..	..	1	21
Totals.	..	..	4	40	178	348	219	91	23	19	6	6	5	1	11	951

were 27 who showed hyperglycemia; but only two such were found among 467 patients younger than 45, an incidence less than ½ of 1 per cent. This simple fact, which is entirely free of any subjective interpretation of emotional reactions, constitutes, in my opinion, strong evidence that hyperglycemia, when encountered in psychotic persons, is a sign of failing function rather than a sign of excess function. If hyperglycemia were a common characteristic of an

excessive emergency activity of the adrenal glands in strong emotional excitement, one would expect to find it occurring in the young and physiologically vigorous group.

TABLE 2.

FREQUENCY DISTRIBUTION OF FASTING BLOOD SUGARS, IN SPECIAL GROUPS.

Blood sugar mg. per cent. / Age.	40-49	50-59	60-69	70-79	80-89	90-99	100-109	110-119	120-129	130-139	140-149	150-159	160-169	170-179	180 and over.	Totals.
Men.																
10-19..	..	..	1	..	4	7	8	1	..	..	..	..	..	..	..	21
20-29..	..	..	..	4	22	32	5	3	..	1	..	..	..	..	..	67
30-39..	..	..	..	4	27	28	17	10	4	..	..	..	..	..	..	90
40-49..	..	..	..	1	15	27	10	1	1	2	1	..	1	..	..	59
50-59..	..	..	..	5	16	31	22	7	..	..	..	..	1	1	2	85
60-69..	..	..	..	2	11	22	17	6	..	1	1	..	..	..	1	61
70-79..	..	..	..	1	6	11	11	..	3	1	..	..	..	..	1	34
80-89..	..	..	..	..	1	5	4	1	..	..	..	1	..	..	..	12
Totals.	..	..	1	17	102	163	94	29	8	5	2	1	2	1	4	429
Women.																
10-19..	..	..	..	4	6	7	3	..	..	..	..	..	..	..	..	20
20-29..	..	..	2	10	17	34	21	7	3	2	1	..	..	..	..	97
30-39..	..	..	..	1	21	44	24	6	1	..	..	..	..	..	1	98
40-49..	..	..	..	3	15	41	25	12	5	4	..	..	..	..	..	105
50-59..	..	..	1	1	7	35	27	20	1	5	1	3	2	..	2	105
60-69..	..	..	..	1	9	13	16	12	5	2	2	2	1	..	2	65
70-79..	..	..	..	2	1	9	7	2	..	1	..	..	..	..	1	23
80-89..	..	..	..	1	..	2	2	3	..	..	..	..	..	..	1	9
Totals.	..	..	3	23	76	185	125	62	15	14	4	5	3	..	7	522
Very excited.	..	..	..	2	4	11	10	3	1	..	1	..	..	..	..	32
Gray's clinical normals	2	6	22	34	59	97	116	66	19	8	..	1	1	..	..	431

Lest undue importance be attached to the hyperglycemics over 45, it should be emphasized that they constitute, even in those ages, a small minority; the great majority have blood sugars within strictly normal limits. As shown in Table 1, where the figures on all patients are given by age-decades, the most frequent finding is a blood sugar in the range of 90 to 99 mg. per cent not only for the

whole group but for each decade separately without exception. The next most frequent range is from 100 to 109, for the whole group and also more especially for those over 45; but in younger patients blood sugars in this range are slightly less frequent than are those from 80 to 89. In the questionably normal zone, from 120 to 139, there are but a few persons (slightly more than 4 per cent of the total) and the frequency after 45 years is about twice as great as among the younger patients.

In consideration of the fact that the dividing line appears at about the time of the menopause, the separation of the data into sexes, as summarized in Table 2, exhibits a tendency of much physiological interest, namely, that the increased frequency of hyperglycemia after 45 (though still a small minority), is definitely more marked among the women. This statistical change appears suddenly near the close of the fifth decade and persists, though with a slight diminution, thereafter. Figures in the questionable range 120-139 mg. per cent show a similar, though less marked accumulation in the decades after the forties.

For comparison, it may be stated here in addition, that the definitely diabetic patients (excluded from the tables) run in a somewhat similar age distribution—one each in the fourth and fifth decades, seven in the sixth decade and four in the seventh decade. It is also significant that among those showing hyperglycemia who were not definitely classed as diabetic, the fasting blood sugar was found repeatedly above 140 mg. per cent for periods of weeks or months in two-thirds of those who were followed through.

The probable inferences from these facts regarding age may reasonably be formulated in the following statement: There is a slight general tendency for the blood sugar level to rise with advancing age, but this general increase is less than 10 per cent, in the whole age range from the second to the ninth decade, as judged by the most frequent findings at different ages. The frequency distribution indicates also that psychotic patients have, in general, normal figures for fasting blood sugar. Hyperglycemia, which is not very common at any age, is almost restricted to the later decades, occurring particularly after the menopause in women, and is usually persistent. These characteristics indicate a closer resemblance to diabetes than to the emotional hyperglycemia of cats.

Diagnostic Groups.

In the diagnostic analysis of the data, I have also excluded the definite diabetics, except where they are specifically mentioned. A considerable portion of the data can be disposed of very briefly because of the rarity of hyperglycemia.

Among 151 classified as manic-depressive manic, of whom 115 were under 45 years, hyperglycemia was observed once. (Case 1. This is the single instance, already noted, which might plausibly be interpreted as an emotional hyperglycemia.)

Among 107 classified as dementia præcox or schizophrenia, of whom 86 were under 45 years, two showed hyperglycemia (Cases 2 and 3).

A miscellaneous collection of 214 persons (of whom 140 were under 45 years) consisted of the following groups: undifferentiated psychosis 39; paranoid condition 34; psychopathic personality 29; manic-depressive mixed 24; psychoneurosis 18; acute alcoholic hallucinosis 15; inebriety 14; not insane, but without further specification 13; drug psychosis 13;* feebleminded 5; epileptic 3; brain tumor 2; traumatic psychosis 2; and one each of Korsakoff's psychosis, exhaustion psychosis, and tabes dorsalis. Of all these, but one showed hyperglycemia—a man of 49, blood sugar 161, who was mildly depressed but classified as "not insane."

From the foregoing figures, which account for 472 persons (very nearly half of our series) no positive conclusion can be drawn beyond the fact that hyperglycemia is rare (0.8 per cent) which is, for the most part, a mere reflection of the facts that most of those here included are young (70 per cent are under 45 years) and that not many of the older persons here included had organic disease. In Gray's collection of 431 "clinical normals," the incidence of hyperglycemia was about 0.5 per cent.

In contrast with these are the manic-depressive depressed (345 persons), the senile and cerebral arteriosclerotic (95 persons), and a collection of other organic psychoses (39 persons). Among these

*Two other persons, provisionally diagnosticated drug psychosis because of the predominant influence of drugs in the production of the clinical picture at time of admission, are otherwise classified here, on the basis of their whole course—one as a psychosis with cerebral arteriosclerosis, and the other as a psychosis with other somatic disease (angina pectoris, Case 6). Both had hyperglycemia.

groups, which are composed predominantly of persons over 45 years, the incidence of hyperglycemia attains significant proportions, amounting in the total to 25 among 479, or 5.2 per cent.

At the lead in this respect is the miscellaneous group of 39 organic conditions (arbitrarily excluding diabetics). Five, or 12.8 per cent, had hyperglycemia, namely: among 20 general paretics, one, a man, 52 years old, whose fasting blood sugar, obtained one morning during a lull in a series of convulsions, contained 199 mg. per cent glucose. (Several months before the onset of convulsions, his blood sugar was 99, and a week after they ceased it was 95.) Among 19 cases classified as "psychosis with other somatic disease," were four who showed hyperglycemia (see Cases 4, 5, 6 and 7).

The groups of senile psychosis and psychosis with cerebral arteriosclerosis are here combined because these conditions were usually combined, but there were 10 who showed clinically only the senile picture. None of the 10 had a fasting blood sugar greater than 120 mg. per cent. Among the 85 others, who showed indications of cerebral arteriosclerosis, were six with blood sugars respectively, of 165, 182, 207, 215, 243, and 276. The incidence of hyperglycemia for the combined senile group is 6.3 per cent; for the cerebral arteriosclerotics separately, 7 per cent.

Under the diagnosis of manic-depressive depressed are grouped 345 patients. (Following Dreyfus, we include here the clinical picture of involution melancholia.) Fourteen had hyperglycemia, all in a mild degree, an incidence of 4 per cent for the whole group, or 6.2 per cent if calculated on the basis of the 224 who were over 45 years old. None of the 121 under 45 years had fasting blood sugar as high as 130 mg. per cent. Although the attempt at differentiation between retarded depressions and agitated depressions is not very satisfactory, one can also say at least, that there were several representatives of both types among those showing hyperglycemia.

It would be fair to say, in a general summary, that the diagnostic groups showing the highest incidence of hyperglycemia are the psychoses with cerebral arteriosclerosis and other somatic disease, and next in order come the depressive reactions of the later decades of life; while persons in other diagnostic groups such as manic-depressive manic and schizophrenia very rarely show hyperglycemia. (This statement should perhaps be guarded by the reminder that

in certain minor groups such as epileptics, whom we seldom receive, the number of cases here reported on, is small.)

The questionably normal blood sugar figures from 120 to 139 mg. per cent show a more scattered distribution through the different diagnostic groups than does frank hyperglycemia, but there is a tendency to accumulate in the same groups as the frank hyperglycemias.

The 13 diabetics who have been excluded from the preceding discussion of diagnostic groups, were distributed as follows: Six manic-depressive depressed and one each, dementia præcox, psychopathic personality, inebriety, paranoid condition, psychosis with cerebral arteriosclerosis, psychosis with other somatic disease (terminal tuberculosis), and psychosis with delirium of unknown origin.

Changes in Mental Condition.

I had hoped at the beginning of this study to demonstrate a relationship between hyperglycemia and strong emotional reactions, and later, when that hope dwindled, to pick up whatever small correlation might be found by a more subtle consideration of mental trends. The net result from the psychological viewpoint is rather small, and extremely simple—that those few psychotic persons who show a hyperglycemia are generally sad, whatever the diagnostic classification, but sadness alone does not produce hyperglycemia. (Incidentally very few of those who knew of their potential or frank diabetes seemed troubled about that. They appeared much more concerned about their ideas of sinfulness or worthlessness.)

Of special psychological interest are nine patients who showed depressive and melancholic reactions (most of them classified as manic-depressive depressed) who had a tendency to persistent hyperglycemia and whose blood sugar was estimated at intervals through the psychosis and into a period of psychiatric improvement or recovery.* Five were frankly diabetic, the other four might be

* The fate of the other hyperglycemics was as follows: Twenty-five showed no significant psychiatric improvement while under observation. (Eight died, eight were transferred to other hospitals, two were discharged and we still have at this hospital seven, of whom two no longer show hyperglycemia.) The other eight were discharged after improvement or recovery. (Three had shown hyperglycemia on only one occasion, and five had approximately the same degree of hyperglycemia, between 140 and 160 mg. per cent, on discharge as on admission.)

considered mild or "potentially" diabetic, though they are not so classified here (see Cases 6, 7, 8, 9, 10, 11, 12, 13 and 14).

All of these cases, when psychiatrically improved or recovered, showed some improvement in the diabetic tendency, as a decrease either in their insulin requirement, or in their necessary food restriction; although in two instances the improvement was slight and perhaps questionable. The association of mental improvement and somatic improvement raises the question as to which was cause and which was effect. I had expected that the administration of insulin or of a better balanced diet might improve the mental condition more especially because the patients were able to maintain or increase body weight, but experience did not justify this expectation. The depression appeared to run its course, unaffected by these aids; yet when the depression cleared the organism required less assistance from without in handling the carbohydrate metabolism.

So far as this evidence goes, it indicates that the depressed mood itself may decrease the capacity to metabolize carbohydrates.

Discussion of the Physiological Implications.

The still common belief that acute emotional reactions in psychotic patients produce hyperglycemia through discharge of epinephrin from the adrenal gland and consequent excess mobilization of glucose, is difficult to maintain on the basis of published communications. The evidence is to some extent conflicting, but mostly negative as collected and summarized by Bowman and Kasanin.[2] The persistence of the belief, in spite of the weight of the evidence, is perhaps attributable largely to two considerations: first, admiration for the beautiful experimental work by Cannon and his collaborators demonstrating the rôle of the adrenal glands in the emotional reactions of cats; and, second, the empirical fact, familiar to psychiatrists, that the urines of psychotic patients frequently give positive reactions when tested for sugar, especially at the time of admission. So familiar a fact, so cleverly explained, renders contrary evidence unpalatable. But in my experience not more than one in a dozen of these apparent glycosurias are attributable to a rise in blood sugar. A few are renal glycosurias, two examples of which were encountered in this series. Scores of them are only pseudo-glycosurias, due to inadequate water intake (which is very common in inadequately supervised patients when manic or de-

pressed) and the consequent concentration in the scanty urine of those reducing substances usually present only in traces too small to affect the sugar reagents ordinarily used.*

A study of the data here reported on 958 patients should assist materially in dispelling beliefs inadequately founded. These observations show the following points of divergence from what would be expected on the emotional-adrenal hypothesis: (1) neither hyperglycemia nor high normal figures were more frequent among very excited patients than among the others, and perhaps no more frequent than among "clinical normals"; (2) hyperglycemia is very uncommon among young patients, whatever their emotional state, and relatively much more common among the elderly patients, especially those with organic disease; (3) about one-third of those showing hyperglycemia are frankly diabetic and in most of the others the hyperglycemia persists with sufficient regularity to suggest rather a limitation in insulin production than a momentary epinephrin discharge; and (4) the teleological conception of the emergency function of adrenal hyperglycemia as an excess provision for violent physical exertion, does not accord at all well with the type of emotional reactions actually observed in patients whose fasting blood sugars are high. They are usually sad and dispirited.

These observations lead me to infer that the physiological mechanism, demonstrable in cats, by which emotional reactions produce hyperglycemia through the mid-brain, sympathetic nervous system, adrenal medulla and liver, is seldom if ever thrown into similarly *excessive* action in the emotional excitement of psychotic patients. Presumably it is the large human cerebral cortex which is responsible for the inhibitory restraint. Surgically decorticated animals † may be considered, on this assumption, as at the opposite extreme, for in them the behavior of strongly emotional type which

* Indeed, in my experience, the common combination of a *slightly positive* Benedict test, in a urine of *high specific gravity* and *strong color* is fairly good evidence against diabetes or hyperglycemia of any origin.

† Although it does not bear directly upon the hyperglycemia-mechanism, it is a fact of considerable interest in this connection that decorticate cats, which show "pseudo-affective reactions" on comparatively slight stimulation, do not persist in this behavior unless the stimulation is continued. (Bard, personal communication.) Speaking generally, in psychotic patients the persistency of affect is most extraordinary.

is so readily elicited, is associated with an exaggerated hyperglycemia, negotiated through the adrenal glands.[6]

It is appropriate to consider at this point the differences in the emotional experiences of cats and patients. As for the inner experiences of the subjects themselves, the observer has no valid means to compare them, either with each other or with his own inner experiences. One is reduced to the necessity of *observing behavior,* and can interpret it only with considerable caution in the light of quite vague impressions and "general knowledge." It is sometimes said that the emotional hyperglycemia of cats is elicited by situations of "real" danger, whereas the emotional excitement of psychotic patients (which is not accompanied by hyperglycemia) is based on imaginary situations. I do not believe that this implied doubt of the genuineness of emotions which are based on imaginary rather than real situations carries much weight with anyone who has examined with care the circumstances of his own emotional experiences and learned thereby the important rôle of the imagination in "real emotions." The contrast does, however, emphasize that for the determination of behavior, great significance must be attached to the comprehension of the meaning of present circumstances derived from individual past experience, and the expectations based thereon. In this respect the human species might well be expected to differ from laboratory animals, for the cerebral cortex is so much more predominant and it is the cerebral cortex which brings these influences to bear upon the organism and thereby modifies emotional experience and behavior.

In addition to the evidence given already, that the hyperglycemia sometimes observed in psychotic states is ordinarily due to inadequate insulin production, rather than excessive adrenal activity, one should consider the results of studies on the demobilization of glucose after feeding it or after administering epinephrin or ephedrin. My own (unpublished) observations are in general agreement with the results reported by others, namely, that a considerable percentage of psychotic patients, particularly those in the depressed mood, show unusual delay in the return of blood sugar to the fasting level. The largest series of observations has been reported by McCowan and Quastel[7] who attempt, rather cautiously, to place their observations in line with Cannon's theories and to explain the sustained hyperglycemia after sugar ingestion as a result of

epinephrin discharge. This is not a satisfactory explanation, unless one makes the additional, and rather pointless, assumption that the epinephrin discharge occurs at the time of the glucose ingestion. Epinephrin, if introduced earlier, has the same stimulating effect on insulin production, as does any hyperglycemia, and thereby produces in the presence of a normal pancreas an *increased* capacity to demobilize glucose.[8]

The facts regarding fasting hyperglycemia, which is comparatively uncommon in any psychotic condition and delayed recovery from a carbohydrate load, which is fairly common in depressed patients, fall more consistently into line with the assumption of a depressed insulinogenic function.* This might be due to a central restraint of the vagus, which controls in some degree the production of insulin. I have been encouraged to make this suggestion by the results of recent studies [9] on the heart rate in emotional reactions, in which we encountered some evidence in the time relationships that the cardiac accelerations associated with brief emotional reactions may be negotiated through central inhibition of the vagus, rather than through sympathetic activity. Incidentally, from the standpoint of its value as a laboratory indicator, the heart rate is a very sensitive indicator of emotions, even when they are slight and brief; whereas the blood sugar has no practical value as an indicator, even in the most extreme emotional excitements that I have observed.

Conclusion.

It seems most consistent with our present knowledge and the observations here reported, to schematize the relationship of carbohydrate metabolism to emotional reactions in psychotic persons, thus: emotional excitement produces rarely, if at all, any rise in the fasting blood sugar. Melancholic reactions appear to depress somewhat the insulinogenic function of the pancreas. Ordinarily this depression of function is demonstrable only as a moderately delayed demobilization of glucose, under a load; so that

*It is difficult to evaluate in terms of normal human physiology the observations reported by Cori and Cori (J. Biol. Chem., 79, 343) who found a decreased utilization of sugar in the peripheral tissues of the rat, when injected with large doses of epinephrin—0.02 mg. per 100 gm. of body weight, which would be in the proportion of 14 cc. of 1:1000 solution for a man weighing 70 kg.

fasting blood sugars fall within normal limits except in those few persons, usually older than 45 years, whose capacity is already somewhat limited by diabetes or other organic disease.

I wish to acknowledge, with thanks, that I am indebted to many for help in this study: to members of the medical staff of this hospital for information about patients and for collecting many specimens and to Mr. C. O. MacCullom and Miss (now Dr.) Jessie R. Cockrill for technical assistance.

BRIEF SUMMARIES OF SPECIAL CASES.

CASE 1.—Mrs. Hbr, married woman of 29, sudden onset of excited, hallucinatory delirium and impulsive violence with brief periods (hours) of calm lucidity and other brief periods (minutes) of suicidal depression.

At venipuncture she was very excited and resistive after a restless, talkative, sleepless night. Blood sugar 148 mg. per cent. Her clinical condition remained about the same for several weeks though she developed a fairly persistent belief that she was pregnant and had some vaguely expressed divine mission. Meantime, on another very excited occasion, her blood sugar was 106. She became gradually quieter, and cooperative, and was discharged after six months as "recovered." Shortly before discharge, while emotionally calm and pleasant, her blood sugar was 95.

CASE 2.—Miss Bdi, unmarried, successful secretary. Confused paranoid onset two months before admission. Sought to escape underworld agents, sometimes by long frantic walks, but stayed fitfully at work. Here she was for some months confused, labile in mood, often erotic in behavior and sarcastic in speech, careless in manner and dress, but did not happen to show any noteworthy emotional reaction at any venipuncture. Blood sugars; four days after admission 186 mg. per cent, a week later 101, on two occasions months later 95 and 104. She gradually deteriorated.

CASE 3.—Mrs. Mz, married woman of 67, quarrelsome paranoid præcox of many years' duration. On one occasion, when sarcastic but not otherwise apparently emotional, her blood sugar was 159 mg. per cent.

CASE 4.—Mr. Cbn, man of 84 yrs., comatose, with severe cardio-vascular-renal disease. Blood sugar 156 mg. per cent. He died the next day of uremia.

CASE 5.—Mrs. Kbl, woman of 57 yrs., delirious, febrile and almost moribund on admission. She lived, however, for 10 days more and was found at post-mortem examination to have had a pyonephrosis. Her first blood sugar was 300 mg. per cent. It rose to 405, but was reduced to 147 by moderate doses of insulin, without apparent clinical benefit. (Possibly she had diabetes, but we did not consider it proven.)

CASE 6.—Mr. Ljy, man of 65. Had at first examination a blood sugar of 181 mg. per cent. He had angina pectoris, had been overloaded with about a dozen different "sedative" drugs and arrived here in a somewhat fearful, delirious state. It required moderate carbohydrate restriction for about a month to keep his fasting blood sugar near normal (the figure running between 130 and 162). He recovered from his delirious reaction with moderately good insight and was gradually put on an unrestricted diet, with resultant fasting blood sugar between 119 and 130.

CASE 7.—Mr. Kz, man of 54 yrs., depressed and anxious, with chronic nephritis, had also a persistent hyperglycemia, varying from 148 to 210 mg. per cent (while the blood nonprotein nitrogen ran from 84 to 147 mg. per cent). Under dietary restriction (of protein) his N. P. N. was brought down to the 40-50 range and his blood sugar then ran between 100 and 118.

CASE 8.—Mr. Gly, man of 54. Manic-depressive depressed. Worried, retarded, fearful of food-poisoning. Blood sugar 154 to 175 on unrestricted diet, brought below 130 by moderate restriction of carbohydrate food. He recovered from his psychosis in six months, and when dietary restrictions were then removed his blood sugar remained at 130 mg. per cent.

CASE 9.—Mrs. Tle, woman of 64 yrs. Manic-depressive depressed. Retarded, nihilistic ideas. Blood sugar at two month intervals 146, 130 and 110. She was not given dietetic or insulin treatment. Discharged after five months, psychiatrically much improved.

CASE 10.—Mr. Fbh, man of 56 yrs. Manic-depressive depressed, and diabetes. Fidgety, slept poorly, depressed, agitated, clear. Blood sugar 221. He had been under treatment for diabetes by moderate dietary restriction of carbohydrates before admission, and when put again on that diet his fasting blood sugar came down promptly to 135 mg. per cent and stayed in that neighborhood until discharged, after about three months, much improved. Eight months later his physician reported to us that the patient's blood sugar remained "within normal limits."

CASE 11.—Mr. Bza, man of 60 yrs. Manic-depressive depressed and diabetes. Fearful of attacks on person and property, agitated and apprehensive, very much confused. Not much concerned over diabetes for which he had been receiving medical advice and treatment (?) for two years. His fasting blood sugar dropped from 220 to 130 when carbohydrates were sharply restricted and remained in that neighborhood for about four months, with gradually increasing liberality in diet. He lost much of his melancholic agitation but remained suspicious and was transferred to another hospital after seven months. Last blood sugar 105.

CASE 12.—Mrs. Frs, woman of 45. Depression and diabetes. Slept poorly. Worried about affairs which were vaguely hinted at. Sometimes vivacious and sometimes quietly sad. At admission on a steady restricted diet and 40 units of insulin daily, her blood sugar was 182. It fluctuated between

190 and 157, as the carbohydrate intake was moderately increased. When discharged, psychiatrically recovered after seven months, her blood sugar was 168, on 33 units of insulin daily and on increased carbohydrate allowance.

CASE 13.—Mrs. Mcy, woman of 53 yrs. Manic-depressive depressed and diabetes. Steadily sad and retarded. Has had diabetes for years but has been careless about diet. Blood sugars first week 253 and 264. With 60-90 units of insulin daily, and a liberal diet, blood sugar was brought down to 185 which was about her threshold. After five months she became less depressed, required only 20 units of insulin daily (blood sugar 187) and was discharged. She returned four months later, again depressed and retarded, with blood sugar 268. Under 40 units insulin daily (blood sugar about 175) she remained steadily depressed for the greater part of seven months and was then discharged as recovered from her psychosis—blood sugar 160, under 30 units of insulin daily. This woman had hypomanic periods outside the hospital, during which she had found that she "could eat anything and as much as she liked" without glycosuria or other diabetic symptoms. This made her skeptical of medical advice and careless about following it.

CASE 14.—Miss Kal, woman of 56. Manic-depressive depressed and diabetes. Inadequate and sad, underweight, irritable, but not specially agitated. Blood sugar 232. Under insulin, 45 units daily on liberal diet (blood sugars about 180) she gained weight but remained depressed for about eight months, then improved rather rapidly and was discharged, her blood sugar being about 140, under 40 units insulin daily.

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DISCUSSION.

ADA HARRIS WALKER, M. D.—Twelve years ago, when Dr. Whitehorn began his studies there was a prevalent opinion, which still lingers to a greater or less degree that hyperglycemia was concomitant with emotional stress.

This, perhaps, was due to a great extent to Dr. Cannon's experiments on cats already mentioned by Dr. Whitehorn. Naturally researches in blood sugar in the various psychotic states followed.

Since that time many contradictory statements are found in the literature, the conclusions in many instances being drawn from relatively few cases in a series, and in the majority the findings are of a negative character as pointed out by Bowman and Kasanin, who reviewed the subject extensively.

In Dr. Whitehorn's series (the largest on record) of nearly one thousand cases hyperglycemia produced by emotional stress occurs in only one. The fasting blood sugar in the remaining cases is within the accepted normal limits.

The tendency to a progressively higher blood sugar in older patients where physical deterioration is present is noted by Dr. Whitehorn and coincides with the studies of Dr. H. J. John of 22,808 cases in the Cleveland Clinic.

In an analysis of 100 cases from my own files recently, I found the average blood sugar was 86 mg. The eight cases between 95 and 120 showed beginning degeneration changes accompanying old age. Six with blood sugar above 120 were unquestionably diabetics, five of whom were over 50 years of age. Four cases in which patients entered hospital for operation with blood sugar between 85 and 95 and were kept under observation on a regulated diet for several days, showed a slight decrease in blood sugar on morning of operation instead of increase as might have been expected if emotions were a causative factor in hyperglycemia.

In a depression psychosis frequently a sustained hyperglycemic curve has been noted and existed in Dr. Whitehorn's series. This raises the question whether metabolic or muscular inactivity is not the cause of hyperglycemia rather than adrenal hyperactivity.

The difference between emotional reaction of a normal and of an insane person is a moot question but Whitehorn's series indicates rather conclusively that the fasting blood sugars in psychotics are within normal limits and hyperglycemia is not produced by emtional stress.

THE NEUROPHYSIOLOGICAL EFFECT OF INTOXICATING DRUGS.*

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(From the Iowa State Psychopathic Hospital.)

In previous experiments we studied a group of patients with psychoneurotic and schizophrenic reaction types by the pharmacodynamic method.[18] A series of intoxicating drugs (sodium amytal, cocain, hashish, mescaline) was administered at certain intervals and the changes in experience and behavior were observed. We found that the typical effect of the drugs, which we knew from observations in normal individuals, was modified in our experiments by the type of pathological reaction which the patients presented. The degree and quality of the alterations in behavior patterns throw new light on the nature of the drug effect as well as upon the psychopathological structure of the different forms of disease which we studied. Sodium amytal for example, which in normal persons furthers the friendly contact with the outside world and decreases self-absorption, showed its effect most markedly in individuals who in schizophrenia had withdrawn their interests from the outside world. Mescaline which facilitates self-absorption showed but a slight increase of original autistic trends in schizophrenic individuals, but led to emotional storms with striking alterations of behavior in psychoneurotic patients.

The present study was undertaken in continuation of this work with two objectives in mind. First, we wanted to check our findings on a large number of patients, and second, we wanted to extend the scope of our work from the purely psychopathological observation to physiological experimentation. We hoped to find some relationship between the alteration of the patient's psychological behavior and changes which we might find in physiological reactions.

The purpose of neurophysiological studies in psychopathology is to obtain information about the somatic changes which accom-

*Read at the eighty-ninth annual meeting of The American Psychiatric Association, Boston, Mass., May 29–June 2, 1933.

pany or are part of the abnormal pattern of behavior. In the early days of psychopathology the idea of research seemed to be the hope of localizing in a certain part of the brain a defect condition which should be responsible for certain deviations from normal behavior.

Since then Gestalt psychology and the psychopathological studies initiated by Meyer and Bleuler and continued extensively by Schilder, have ascertained that a large part of abnormal behavior patterns is not the result of defect, but the result of efforts at restitution on the part of the whole organism. Abnormal behavior is a pattern of psycho-biological acts, different aspects of which may be shown in physiological and psychological studies, but which cannot be divided into two separate compartments of research. On this basis it was quite faulty to assume, for instance, that any state of stupor would necessarily be the expression of more or less absence or vagueness of inner experience or of a low level of brain activity. While on the other hand marked motor excitement does not allow conclusions about the amount of processes going on in the brain.

Physiological behavior, the same as mental behavior, progresses in a series of patterns. To determine variations of one point in this pattern without due consideration for changes in other related parts of the pattern will lead to contradictory results and faulty conclusions. What we call stimulus is a change in the outer world which usually is followed by the transformation in the behavior pattern which we are studying. In performing laboratory experiments, we confront our patients with a new situation to which they will react in a certain way. Changes in the mental behavior, such as a sudden attack upon the examiner, can be understood only on the background of other mental manifestations such as the affective state. If we carry this principle into the physiological studies, we must look for a *number* of indicators to make demonstrable the pattern of physiological behavior in different sectors of the organism.

It was not difficult to find indicators in the field of metabolism and blood condition; pulse rate, blood pressure, the level of leucocytes, and the blood sugar level could easily serve as indicators for the drug effect. The effect of some drugs in this respect is known. Marked changes in blood sugar level, for instance, were observed by a number of experimenters to follow the injection of adrenalin[8]

and the administration of hashish.[5] Changes in leucocyte level following adrenalin injection were described recently by Martin.[21]

Moreover, reliable and well established methods are available for these determinations. In our study, we used the usual clinical method of determining the white blood count and the method of Janney and Isaacson [16] to determine the blood sugar.

Much more difficulty was encountered when we tried to find a reliable, well established indicator of alterations in neurophysiological processes. Sensory tests could not well be used because of the poor cooperation which a large number of our patients presented. The tests which refer to the motor system are few and most of them the subject of serious controversy between different workers. The only methods which seemed to be fairly well known and to offer the possibility of comparing our results with a wide field of data reported by other observers were the action current method of determining changes in reflex latency periods, the chronaxia method of determining the time excitability of the neuromuscular apparatus, and the method of determining changes in skin resistance which accompany certain alterations in behavior.

All three of the methods have shortcomings. The last method finally had to be discarded because the restlessness of some of the subjects invalidated our findings. The patellar tendon reflex studies have been well standardized by several investigators, particularly L. E. Travis,[27] who developed a fairly high level of accuracy. The chronaxia method is of great theoretical interest and seems to be particularly adapted to give us insight into physiological changes corresponding to psychopathological conditions.

The method was developed in the physiological laboratory [17] on the basis of the fact that tissue excitability can be circumscribed adequately only if we use two parameters, intensity and *duration.* The time factor, so important in behavior, was recognized in its importance for the stimulus situation.

By applying this method to tissues within the whole organism, Lapicque [17, 6] was able to show typical ratios of responsiveness between different functional units, in close relationship to the interplay of synergic or antagonistic biological activities.

It became evident that a determination of only one value was misleading; the chronaxie, for instance, of the biceps muscle was found to increase or decrease in a way which could not be under-

stood unless one took into account the relationship of this measure to corresponding changes of excitability in the antagonistic triceps.

If we attempt to use this method for our purposes, we unfortunately are confronted with the great controversy of physiologists [23, 13, 17] about the significance of the fundamental findings of Lapicque, and moreover, we have to face particular technical difficulties since the method involves, and is dependent to a large extent upon, the accuracy of observation in a given examiner. We were quite aware of these difficulties and tried to overcome them by a number of precautions.

From a theoretical point of view we felt much encouraged since we learned about experiments which have been carried on in Germany (Altenburger)[3] in order to determine the time-excitability-curves of sensory organs for *adequate* stimuli. The results were essentially the same as those found after the inadequate stimulation by electrical currents which we are using.

The condensor method of Lapicque and Stein was used for our purpose. We carefully followed their method of observation. Our findings are open to the same criticisms that have been raised against their work, but since our findings agree with a number of other observations and fit well into the total picture of observed alterations of behavior patterns, we felt justified in reporting them as helpful pointers for further research in a new direction. If our results prove to be valid by further work, they will furnish important evidence for the close correspondence of psychological and physiological reactions, both of which, in our opinion, are only aspects of one and the same biological behavior pattern. If one or the other method which we employed should later be proved to be questionable in the light of new work, we still will have the rest of the picture in our pattern to give a less complete but nevertheless instructive picture of physiological changes.

Method and Equipment.

Ten patients were used for this study; the diagnosis in three of them was psychoneurosis, in seven of them, schizophrenia. They all had been in the hospital for several months and their backgrounds and life situations were fairly well known.

The methods employed were the following:

In addition to the routine physical examination, the patient's sugar tolerance curve was determined, using the method of Janney and Isaacson.[16] With the same method during the subsequent experiments we determined the changes in the blood sugar level resulting from different drugs. In the same manner we studied the changes in the leucocyte count and added observations with reference to blood pressure, heart rate, and the general neurological condition. The special neurophysiological investigations were carried out with the help of a chronaxia apparatus similar to that used by Lapicque[17] and Stein.[28] The apparatus was developed in our laboratory by Jasper[15] and later reported by us in a previous paper.[10] A similar apparatus was employed in this country by Wechsler.[29] A brief account must suffice here.

The method provides quantitative electrical stimuli and allows us to study the responsiveness of the neuromuscular apparatus in a way much more conclusive than was made possible by older methods of electrical stimulation. This advance was brought about by introducing in addition to the threshold for the intensity of a continuous current (for instance, the galvanic current) a second parameter in the form of the minimal duration of a current of double threshold intensity which just produced response.

With this method variations were found in the responsiveness of nerves and muscles, which it was impossible to demonstrate with the older methods. Particularly important for psychopathology was the discovery that muscle groups serving as antagonists in motor coordination usually show a definite ratio of responsiveness under normal circumstances, this ratio being about one to two, the smaller values applying to the flexors of the upper extremities and to the extensors of the lower extremities.

In addition to this there was found a possibility of demonstrating changes in responsiveness within the cerebro-spinal nervous system, following mechanical or chemical stimulation of the vegetative nervous system.

Finally, the great importance of studying any peripheral unit with proper consideration of its place in the whole of the organism became more and more manifest. Muscles and nerves which have the well defined ratio of responsiveness mentioned above under normal conditions, lose this ratio and show marked changes in

chronaxia if they are severed from their proper connection with the body.[17] The "chronaxie de subordination" changes into the "chronaxie de constitution." That all these observations, which originally were made in the physiological laboratory, hold for human and for clinical conditions was proven by a number of authors who showed consistent change of chronaxia, loss of ratio, and fixedness of the values in cases of neurological lesions of the pyramidal type, as well as in extra-pyramidal conditions. In addition to this, the effect of certain toxic substances, particularly of alcohol, was studied and we were able to show in a recent study, loss of ratio and other changes in chronaxia in normal individuals during alcoholic intoxication.

The actual procedure in determining the motor responsiveness is as follows: The response is first determined in the usual galvanic method. The voltage (rheobase) which is necessary to produce a muscle twitch is measured by a rheostat. Condensers of different capacity are then charged with the current of an intensity twice as large as the threshold intensity and then are discharged by a double-rocker switch into the organism. Since the time of discharge of condensers within certain limits is proportional to their capacity, the measurement of the threshold capacity in terms of microfarada can by calculation be easily computed in terms of time (chronaxia).

The patient was placed in a comfortable chair, the indifferent electrode was placed upon his chest, and the other electrode was placed at the point of optimal response of the muscle which was studied. The city current rectified, stepped up to 180 volts, and controlled by a rheostat, was used as stimulus through charging and discharging a very large condenser which supplies this current in practically unlimited duration. A series of stimuli was applied in regular sequence at intervals of about a second by an automatic device which activated the rocker switch. The minimal intensity just producing a muscular response was recorded as "rheobase." It then was doubled and the calibrated bank of condensers was decreased so much that the response just disappeared. The time value calculated from the capacities found in this way were in a range of from one ten-thousandth to a thousandth of a second. With this procedure we found in normal individuals a marked consistency of the threshold of responsiveness through

days, independent of the position in the state of tonus of the motor apparatus. This is in accordance with the findings of other writers.

In order to do away with the uncertainty which necessarily is connected with the subjective factor in judging a minimal twitch of muscles, we developed a recording device which allows the marking of the extent of the muscle response on a polygraphic record, together with an indication of the condenser combination which was used for a given moment. The method is the following:

The motion of the muscle is transmitted to a small lever system which moves a tiny secondary coil in the field of a pair of primary coils. The alternating city current, reduced to four volts, is sent through this primary coil, furnishing induction current in the secondary coil of an intensity which varies with its position in the magnetic field. The changes in intensity of the secondary current are amplified by a usual commercial amplifier which activates the magnet of a polygraph writing pen. By this method it is possible to compare twitches of equal size with each other, determining chronaxia values belonging to a previously determined rheobase.

The value of this method in physiological or pathophysiological studies in humans was doubled lately on the basis of recent investigations by Rushton [23] and Grundfest.[13] The latter found that Lapicque's claim, that the size of the electrode is of little significance for chronaxia results within certain limits, does not hold. He found that different chronaxias, or as he called them "excitation times," applied to different tissues, and he states, that since in experiments with humans the electrode never can be applied directly to nerves or muscles, no evidence is available to tell us the area of excitable tissue which is actually exposed to the stimulating current. This would produce the danger of using chronaxia values which belong to a different rheobase than one assumes. Lengthy and complicated discussion has developed upon this point. For our purposes the most important demand is that we control our findings as carefully as possible. Since we are not intending to find absolute values but only relationships within one and the same organism, the minute variations which might result from the difficulty which Rushton showed, will be of very little importance; nevertheless, we followed the suggestion of Achelis [1] to follow the chronaxia determination by a second determination with

a rheobase of 10 volts higher than the original one and to use the value only if the two findings have a proper relationship.

The reflex time latency period was determined by the action current method developed by Travis.[27] The patient was placed in a comfortable chair, and his patellar tendon was stimulated by a stroke with a hammer. The stroke produces a signal on a recording film of an oscillograph; the other units of this oscillograph are connected with electrodes which furnish, through a three stage amplifier, the minute changes in electrical potential which, so-called action currents, accompany the activity of the quadriceps muscle subsequent to tapping the tendon. The action currents appear after a certain latency period, which, by Dr. Travis, has been found to have marked consistency in normal individuals while under the influence of alcohol, and in certain cases of mental disease there was a change in this time. We recorded the reflex latency period under the influence of different drugs in our patients three or four times in order to study the consistency and the direction of deviation.

The third method from which we had hoped certain results, namely, the determination of changes in skin resistance by psychogalvanometric studies during the presentation of a moving picture to the subject, did not give sufficiently satisfactory results. So far it has been impossible to control the patient's movements in the dark room to a sufficient degree.

The experiments were usually carried out in the evening between eight and midnight. The patient, who had not received any food since noon, was taken to the laboratory, placed in a comfortable chair, the situation was explained to him, and his initial reactions were noted. The neurological condition was examined. His blood sugar level and leucocyte count were determined, the chronaxia values of the four muscles of the upper right extremity were recorded, and the patellar tendon reflex latency period was ascertained. The patient then received the drug and at intervals of from 15 to 30 minutes subsequent observations were made until after from three to four hours the changes seemed to be recorded sufficiently. A free interval from 36 to 48 hours was allowed to elapse before the next experiment was started.

The amount of drug to be administered was determined relative to the weight of the patient. All the drugs were administered by

injection except cannabis indica, which was available only in the form of the extract and had to be given by mouth.

We were able to carry through our series of experiments with 10 patients. Hashish was given to only four because of the uncertainty of dosage. The chronaxias and blood studies were carried out in all of the patients. The determination of the reflex time was limited to six of them.

Psychological Observations Under the Influence of the Drugs.

The following is an account of the behavior of our subjects during the experimental periods:

Subject I.—L. D., a 47-year-old business man, admitted because he felt fatigue, restlessness, believed himself to be doomed to permanent invalidism, had numerous distressing sensations in his stomach region, and vague headache. The condition had developed in his adolescence when he learned about the alleged serious results of masturbation. His sexual adjustment was quite unsatisfactory. He did not marry, lived with his brother, gradually gave up his business activities, and during the last years has spent most of the time in bed or resting, more and more absorbed in his hypochondriacal thoughts. His physical condition was found to be quite good. All laboratory findings were within normal limits. He was of decidedly asthenic build. His behavior in the ward was that of a person who is convinced of permanent invalidism, but there were no somatic delusions. Reassurance about the nature and rôle of masturbation would only temporarily change his attitude. It was not possible to win sufficient emotional rapport with him to find important material in the nature of early experiences.

On March 23, 1933, he received 1 ccm. of adrenalin subcutaneously. His account of his experiences was not different than at any previous time. He claimed there was no change whatsoever in his feelings. His outward behavior did not show any change.

On March 27, 1933, he received 4.5 grains of sodium amytal intravenously. Having been rather reticent and unwilling to communicate important facts previously, the patient became more cheerful, fairly talkative, described feelings of exhilaration, and seemed serene but somewhat sedate, dignified, as if enjoying the freedom from distress, without, however, much reaching out for contact. The effect ceased in about two hours and the patient became sleepy.

On March 29, 1933, injection of 30 mgm. of cocaine. Behavior and experience showed practically no change except mild feelings of exhilaration and restlessness. His stomach trouble was not as depressing as usual. He felt contented and had an air of cooperativeness, although he did not give up his reserve.

On April 2, 1933, .2 gm. of mescaline subcutaneously at 10.20 a. m. At 11.00 dilation of the pupils. Restlessness. At 11.20 the second injection of .3 gm. of mescaline. A few minutes later a marked increase of restlessness, flash-like twitches of the muscles of the cheek, marked rhythmical contraction of the muscles of the lower extremities, pupils markedly dilated. Patient is quite uncommunicative. He emphatically states that there is no change in anything; yet at the same time his body is shaken by hiccough-like movements, his head jerks backward, and he trembles severely. The patient states he has no feelings of spontaneity in connection with these jerking movements. He describes them as involuntary. Upon closing his eyes, he does not have any visual impressions. At 11.50 there is marked difference of the pupils and continued motor restlessness. There is a motor over-reaction to sensory stimuli. A touch with the hair bristle produces a marked jerking. He grits his teeth and holds his arms tight to his body. He complains about feeling sick, as though his nerves are going to pieces and as if the nerves were over-charged with electricity. He complained about marked weakness in his stomach, "as though he could feel his stomach lying in his body."

SUBJECT II.—E. L., a 22-year-old boy, admitted because of heart palpitations, feelings of gastro-intestinal distress, light-headedness, tendency to dizziness, and a marked feeling of vague fears without object. This condition had developed in 1931 in connection with worries over masturbation. Reassurance on the side of his home physician did not succeed in dispelling his anxiety state. The patient was a well-developed young man of athletic build, who, at the time of his admission, presented the picture of a marked anxiety neurosis. In the course of psychotherapeutic efforts early experiences were uncovered which had conditioned his fear. Homosexual experiences in adolescence with older men had revived an earlier ambivalent attitude towards his father and masturbatory practices were carried on in close association to homosexual daydreaming. Under the influence of treatment the patient had improved markedly at the time at which our experiments were carried on. Being still somewhat anxious and easily perturbed, he had lost his physical symptoms and was taking a normal interest in the activities in the hospital. The physical examination had been negative.

On March 30, 1933, the patient was given at 8.40, 1.3 ccm. of adrenalin. His behavior did not change. He did not report any differences in his experiences. His way of communicating was not changed from his usual manner of conversation. There were no neurological signs.

On March 29, 1933, 30 mgm. of cocaine at 7.43 p. m. At 8 o'clock there was a marked dilatation of the pupils, a certain amount of restlessness and gaiety. The patient paced the floor in the laboratory and joked. No other changes were noted.

On April 2, 1933, the patient was given .5 gm. of mescaline at 10.30 a. m. At 10.40 he complained about weakness and dizziness and marked nervousness. He becomes more and more reluctant to talk, seems to be bashful and uneasy. The patient sits hiding his head in his hands, absorbed in his own

experiences. From time to time he sighs deeply. At 11 o'clock the pupils were dilated, and the face is flushed. The pulse is 96. "You all seem so far away." Patient tries to speak about his experiences but has marked blocking. He starts sentences and breaks off without completing them. He sits quiet and listless for long periods of time without answering questions. He lies down on the couch and hides his head in the blanket. Patient continues to be unable to answer questions, and makes no communications about past experiences.

Under the influence of sodium amytal, which he received at 9.05, April 27, 1933 (4.5 gr.), patient feels serene. He smiles a good deal. He turns to the persons around and talks freely about his life. Patient remains hilarious for the next two hours until he goes to sleep.

SUBJECT III.—S. S., a 45-year-old married woman, admitted because of paralysis of the right side, concentric contraction of the field of vision, monocular diplopia. She was treated by suggestion. In hypnotic catharsis she revealed important factors in her childhood which contributed to her present reaction. Her mother had died shortly before the development of her illness and had had a right-sided paralysis resulting from an apoplexy. The patient's symptoms had entirely disappeared at the time of the experiments and she had no complaints.

She was given 6 gr. of sodium amytal on March 20, 1933, at 9.30 o'clock. The patient became talkative, had a nystagmus, some blurring of vision. She speaks quite freely about matters which she had not cared to mention much before. She remembers in detail about the previous hypnosis, which had been covered by amnesia. She becomes quite emotional while reporting this. She talks so fast that she almost stumbles over her words. There is no change in her postural reflexes. There is reduction of responsiveness to touch stimuli. The analgesia of the right upper extremity reappears, also the tremor and weakness. She is very open and affectionate in her remarks and wants to be assured that she is cooperating and she encourages other persons around to speak freely to the doctors. Patient gradually goes to sleep. Until she is sound asleep the tremor of her right upper extremity continues.

On March 27, 1933, at 7.38 p. m., she received 0.25 gm. of mescaline. Ten minutes later she complains about dryness in her throat and nausea, a distress in her stomach, like cramps. She hides her head in her hands, seems afraid and unwilling to communicate. She has a marked dilatation of pupils. When asked questions, she is reluctant to answer and rather irritated. She is afraid that her secrets will not be kept. The anesthesis and paralysis of her right side return. She feels that the floor is too near to her, as if it were raised. She trembles and shivers. She turns away from examiner in order to prevent having to answer questions. At 8.15 she begins to see colors. She sees a change in the atmosphere, as if smoke were in the room. The lights seem "terribly bright." It scares her. The hands of persons around her seem too big. The lights seem to flicker and change in intensity. Objects around her seem to move. If her leg is touched by a pencil, it seems to penetrate the flesh. She begins to speak about visual impressions, about prehistoric animals. "Beautiful flowers and wild animals are mixed up in one mass."

During all this time patient appeared restless, overwhelmed by the experiences, apprehensive, and shy. There was no change in reflexes.

In retrospect, after the disappearance of her symptoms, she told the examiner that during her intoxication she had seen prehistoric animals approaching her to bite her in her neck, that she had become aware that one animal had the eyes of her father, represented her father, and that vividly she was reminded of a scene in her girlhood when her father, when drunk, requested incestuous relations with her. She was aware of the symbolic meaning of the pictures she saw.

On March 23, 1933, the patient received 1 ccm. of adrenalin at 8.20. After injection of adrenalin no psychological changes were noted.

On March 24, 1933, she received 30 mgm. of cocaine subcutaneously at 8.10. Her pulse changed from 70 to 120. She complained about dryness in her mouth, had a marked dilatation of the pupils. She complained about abdominal cramps, about heart pounding, restlessness, and uneasiness. She becomes talkative, feels "quivery," scolds about the doctors in an aggressive and demanding way. She makes jokes, tells examiner that she gets a "big kick out of this all," and tries to razz him about his experimental procedure. She repeats the same statement in a garrulous fashion frequently. Her extremities show no change. Her functional symptoms do not return.

Subject IV.—V. W., a 21-year-old farmer's boy, admitted because of periods of irrational talk and impulsive outbursts against members of his family, which had developed in the last months. They were preceded during the last two years by a state of profound lack in interest and initiative. His illness started after he had made an attempt at working on a farm in a neighboring community. He there formed an attachment to the young daughter of a minister. Her father interfered with her friendship. The patient then developed a state of agitation in which he heard this minister's voice talking to him through the air, warning and accusing him. He returned home, seemed absorbed, quiet, and had outbursts of restlessness. This state gradually subsided and gave way to the defect condition described above. In the hospital, patient shows little interest in happenings around him and does not initiate any conversion or activities. He is superficially polite and can be drawn into activity by others. His answers are brief and usually accompanied by an empty smile. His physical condition and neurological condition were normal. His laboratory findings were within normal limits.

On April 29, 1933, he received 6 grs. of sodium amytal. His behavior and interest in communication do not change. He smiles continually. There is a definite nystagmus.

On April 3, 1933, he was given .5 ccm. of mescaline. During the experimental period, from 8.15 p. m. to 11.00, he showed not the slightest change in his behavior and did not report any change in his experience.

The injection of 1 ccm. of adrenalin on May 2, produced no change in behavior and experience.

On May 9, 1933, the patient received, at 8.55 p. m., 30 mgm. of cocaine. His mental condition shows no change.

SUBJECT V.—M. H., a 34-year-old single woman, admitted at her own request. The family had noticed since August, 1932, an increased self-absorption. She also had mentioned the belief that certain men were interested in her. In the hospital she sat around for hours absorbed in her own thoughts, had a decided lack of interest in things going on around her, and soon, although reluctantly, admitted that she heard the voice of a doctor in the hospital talking to her through the air and over the radio. She did not make an effort at any logical arguments on the subject, but began to think that this physician's child really was her own child. No statements of any kind provoked an emotional response. She is of the dysplastic type. There were no physical findings. The basal metabolic rate was normal.

On March 28, 1933, she was given at 8.20 p. m., 4.5 grs. of sodium amytal. She developed a nystagmus. Her gait became ataxic. Her emotional state is changed. She shows less self-absorption and is more communicative than usual. Her blood pressure shows no change. She says she feels as if she would like to talk a lot but "I'm so sleepy I would like to go to sleep too. I suppose this hospital is a good place to test my nerves."

On March 30, 1933, she received at 9.50 p. m., .25 gm. of mescaline, which was repeated an hour later. She had entered the experiment in her usual placid mood, without showing any interest in her environment. After 15 minutes she smiles a good deal to herself. When she is questioned about it a little later, she breaks out with some verbigerations. "My body feels different. It feels, I seem to have a feeling, feels like, I feel prickling. I feel numb. (Walks.) I feel like, like I was light in weight. It's just lightness, just feels, I feel big, too." This inability to express her experiences and seeking for words and the perseveration of certain words continues. She feels that her hands become light, that her body begins to float. She begins to see the room filled with blue smoke. After about an hour, patient states with a mixture of contentment and surprise, "Everything is burning up. More and more flames are coming. All of them are blue. Everything is on fire. The door is getting black from the ashes." Patient is quite absorbed in her experiences. When she is asked to move around, she steps carefully because she feels she is stepping on a beautiful, deep carpet like a Persian rug in many colors. She feels that she herself is on fire. She wants somebody to take her out of the room. She feels that she is shaking markedly, although there is no objective evidence of it. In retrospect she later speaks of her inner fires, her fire of passion for the physician. She thought if the doctor would come he would protect her and the fire couldn't hurt her.

On April 6, 1933, she received 15 drops of hashish extract at 8.15 p. m. Fifteen minutes later she had dryness of her mouth, dilatation of her pupils. Her usual complacent, smiling attitude remains as it was. She describes again a marked increase in the apparent size of her own body while everybody else around looks about as tall as usual. Objects seem to be in the far distance. She is feeling pleasant and contented. The size and shape of objects around is not consistent. The changes are reported without any sign of emotion or interest. There is no change in reflex activity or any other mood abnormalities.

On April 11, 1933, she was given 50 mgm. of cocaine at 9.20 p. m. She remains quiet, sitting with a smiling expression, speaking rather little, at times appearing somewhat restless.

On April 12, 1933, she received 1.5 ccm. of adrenalin. No change is noted.

SUBJECT VI.—M. S., a 30-year-old school teacher, admitted because since Christmas of 1932, following influenza, she had talked irrationally, begun to imagine that she was the devil and her parents strangers, developed a state of excitement with bizarre utterances and postures. She heard her parents talking through the air and whispered to herself. Her attitude was changeable; at times she talked in an incoherent fashion and at other times she seemed to be in good rapport and made clear statements. She had periods of excitement, attacked members of her family in an impulsive fashion. On admission she was somewhat apprehensive, restless, preoccupied with her own thoughts. She showed blocking in her conversation, was quite evasive in her answers, but was fully oriented and showed no signs of true confusion or fogging of consciousness. Her physical condition was normal. When the experiment was started she showed a rather childish resistance, fighting off in a playful manner those who wanted to take her to the laboratory.

On March 20, 1933, she received 4.5 grs. of sodium amytal intravenously at 9.30 p. m. Under the influence of the drug her reticence and unwillingness to talk changed into marked talkativeness. She shows rather peculiar smiling and chuckling without letting us know why, but seems to feel jolly and much less hostile than usual. She notices that people seem to resemble each other. "Everywhere I go I see somebody that looks like somebody else. The resemblance is so strong that I think they are the same but maybe this comes from the nervous breakdown." "But those things are personal affairs. My thoughts are blocking. Blocking, that's blocking, blocking thoughts." There is no change in her motor systems and no evident change in her experiences.

On March 22, 1933, the patient received at 7.35 p. m., .3 gm. of mescaline. She first is rather talkative but soon shows a good deal of tension and anger. There are outbursts of laughter. She is unwilling to answer any questions, repeating that she wants to be left alone. At 8.33 there is a marked enlargement of the pupils. She complains about headache. Her stream of talk becomes less and less coherent and develops into a regular "salad of words" which she carries on in a sort of sing-song fashion. It is as if she were talking to herself, without any reference to outsiders.

Under the influence of cocaine, 30 mgm. of which was given at 8 o'clock on March 24, she developed a state of marked excitement, talking continually in a loud, singing voice. She misinterprets persons around her, brings in numerous reminiscences, but in such an incoherent fashion that one can't understand them clearly. She speaks about killing "all the single girls and all the single fellows."

On March 23, 1933, adrenalin injection at 8.05. There is no change in blood pressure and pulse rate. By 8.33 the patient mumbles to herself but

one is unable to hear what she says. No appreciable change occurs in her reactions during the experiment.

On April 11, 1933, she received 15 drops of hashish. She becomes rather cheerful, tells funny stories with some incoherence.

SUBJECT VII.—M. P. Patient is 25 years old, unmarried and was admitted because for the last two years she showed a change in personality, had talked to strangers as if they were her friends, dressed herself in a bizarre manner, and began to spread stories about moral delinquencies of ministers and other socially prominent people. She would approach strangers and mumble to herself as if she were talking about them. She became restless, irritable, and began to hear voices talking to her. Even in her high school days she was observed occasionally sitting for long periods of time in a rigid position and staring in front of her. On admission she showed varying behavior, at times friendly and cooperative, at other times having violent, impulsive outbursts. She occupied herself by making numerous drawings, having symbolic content, which gave evidence of her belief in the omnipotence of thought. She was creating new languages, was predicting the course of the world, and during her stay here one could see from day to day the breaking down of her logical reasoning and an increasing withdrawal from the world of reality. Her physical condition was normal. At the time of the experiments she was quite resistive and seemed to have a particular antagonism against the examiner.

On March 28, 1933, she was given 4.5 grs. of sodium amytal intravenously. Her hostile attitude changed immediately to pleasant, friendly conversation. She apologizes for her antagonistic attitude, speaks in symbolic formations of the "union of the heart," and emphasizes that examiner seems to be overworking so far as she can see. She continues to use rather stilted phrases and vague concepts, but is much more coherent than before the medication.

On March 30, 1933, injection of .25 gm., at 9.00, of mescaline. At the onset of the experiment the patient is fairly friendly. After about 10 minutes she begins to be self-occupied. She becomes more and more irritable and antagonistic. She becomes uncooperative during the experiment. Her stream of talk gradually turns into a tirade of scolding. Her language becomes more and more profane and disintegrates into an unending series of cursing and scolding words. With all this, patient does not seem to wait for any effect upon the object of her scolding, but seems to have more a marked affect discharge without much concern about the reaction of others.

On April 11, 1933, patient received 40 mgm. of cocaine. She, since the mescaline experiment, had apologized for her anger and stated that she did not dislike the examiner as much as she had indicated. Under the influence of the cocaine she becomes quite euphoric and assumes a sort of philosophical air. She plays with different types of language, with fragments of poems, sings songs, imitates bird calls and the voices of different animals, dances around in marked excitement. The excitement subsides within two hours.

SUBJECT VIII.—G. F., a 42-year-old, unmarried woman, admitted because she had annoyed people around her by bizarre statements. She claimed that a detective system was developed in order to spy on her, to discover her thoughts, and to do her harm. The condition developed very insidiously during about four years. She believed that millions were being spent to carry out the system of persecution. She had auditory hallucinations and when she is thinking she has the feeling that her thoughts are drawn away by a telepathic device. Here previous personality had been that of a sensitive, retiring, but ambitious person. She always had been prudish about sexual matters, never had any love affairs, and never considered marriage. Her physical condition was quite normal, her build was of the athletic type.

On April 28, 1933, she received 6 grs. of sodium amytal at 9.20 p. m. She was fairly cooperative but slightly sarcastic when she was taken to the laboratory. Under the influence of the drug, she becomes more communicative, laughs a good deal, dances around in a joking fashing, "I feel like a ship on the ocean." There is a nystagmus and poor eye muscle coordination.

The patient received 40 mgm. of cocaine on May 1, 1933, at 9.30 p. m., being fairly cooperative. She seems to be restless but restrains herself. She seems to be ill at ease, tries to laugh the matter off. She becomes antagonistic. She is angry, yet fairly cheerful.

On May 6, 1933, this patient received 1.4 ccm. of adrenalin. The patient becomes more antagonistic during the communication.

On May 2, 1933, the patient received .5 gm. of mescaline at 9.00 p. m., blood pressure 110/55, pulse 84. At 9.15 patient becomes restless, feels nauseated, states emphatically that she is just the same as ever in her mind, develops some shaking of the lower extremities. She has marked dilation of her pupils, states that she is light-headed, things look hazy, and she claims that she has nothing but a bilious attack, such as she used to have before. At 10.55 she felt better, after having been sitting silent for more than half an hour.

SUBJECT IX.—M. E., a 40-year-old wife of a farmer. She was admitted because of anxiety states, restlessness, irritability with her children, and rather bizarre statements in which she spoke about the presence of a man when nobody was around. She would stay in her room, not come down when the neighbors came to visit, became careless about her housework, lost more and more interest in her previous life activities. All the symptoms first were entirely those of an anxiety state, but during the last year they shifted into a state of complacency lacking in emotional warmth. The childhood of this woman had been very unhappy. Her father had been inclined to temper outbursts, domineering. He refused to permit marriage to a man whom the patient loved but forced her marriage to a husband who did not give her any satisfaction. Nevertheless, she had had five children, and it was while bearing the sixth child that she broke down her previous standards, and initiated a miscarriage, having marked feelings of guilt about it afterwards. In the hospital her behavior was changeable. At times she sat around for hours with a contented expression, having a marked lack of interest in

things around her. At times she would be irritable, easily provoked. She showed some blocking in her conversation. At other periods she was willing at least to give a partial account of her life history.

On April 28, 1933, she received 6 grs. of sodium amytal at 9.00 p. m. Three minutes later she begins to laugh. Turning to the examiner she states, "You look different to me than you used to do, much more friendly. I felt hostile to you but I can't tell you why. It would be pretty hard to do that. All the worry and pressure seems to be gone. You know it seems I was afraid of everything. I was afraid to speak to people. What do you want me to tell you?" The patient is apparently quite gay and fairly communicative.

On May 1, 1933, she received cocaine at 9.15. At first she becomes restless, is aggressive and unwilling to talk. She states in an indignant manner that she wants to be left alone, she did not come down here to make a fool of herself. She is suspicious, feels that a session is arranged to spy on her life. The patient becomes more and more irritated. She can't sit still. After 30 minutes she suddenly dashes out of the room and runs with great speed through the hallway and tries to get out of the building. When asked to return, she is quite resistive at first, but finally surrenders and comes back. Her pulse had changed from 82 to 140. Her blood pressure increased from 105/55 to 120/80. The pupils are markedly dilated. She complains about dryness in her mouth.

On May 2, 1933, the patient received .4 gm. of mescaline at 8.50. She states that she feels funny, "sort of sick like." She later is nauseated. Her pupils are markedly dilated. She has an emesis. She is very quiet, self-absorbed. She states that she is very contented. She does not want to speak of any of her experiences. At 10.15 she emphatically states that she feels perfectly all right.

On May 6, 1933, this patient received 1.2 ccm. of adrenalin at 8.10. After 20 minutes she complains about restlessness and shakiness. She has no evidence of motor disturbances and complains about fear and anxiety. The mental attitude otherwise shows no change. The pulse rate increased from 80 to 120.

SUBJECT X.—E. H., a 27-year-old banking clerk who was admitted after he had sent numerous letters to authorities in which he suggested a reorganization of the government and the structure of the state in a very bizarre fashion, adding innumerable symbolic drawings. He had done this the last two years until he had become conspicuous enough to be sent for examination. We found him to be the only son in the family where the father had died two and one-half years ago. There was no tendency for mental disease in the family, but the father was rather quiet and autistic in his thinking. Six years ago the patient went for a year to college and had to take part in the military training. While exercising on the rifle team, he developed a state of anxiety, had to drop his activities, and was sent to a general hospital on the point of a "nervous breakdown" because of shaking and trembling. The period of marked disturbance subsided in a few weeks. Since then the patient has shown less

interest in things around him and busied himself in developing schemes for reorganizing the world. On admission he was found to be of asthenic build without any signs of physical disease. He was polite and cooperative, very shallow in his affective responses. He continued to make his drawings, expressed his belief in telepathy, and told the examiner that his present condition resulted from the negative suggestions of the clergyman who had influenced him to give up marriage and resort to a life of Christ-like purity. He was cooperative during the experiments.

On April 29, 1933, he received 6 grs. of sodium amytal. His mental state showed practically no change except for a tendency to sleepiness.

On May 3, 1933, at 8.55 p. m., injection of .4 gm. of mescaline; at 9.50, .2 gm. After half an hour, patient begins to appear self-absorbed and in a trance-like state. There occur waves of tremor of the lower extremities. The patient doesn't answer questions except for the occasional effort at formulating a sentence or asking, with a helpless expression, the question, "What do you want to know, doctor?" There is nothing to say." After 50 minutes his blood pressure has risen from 110/55 to 145/55 and his pulse rate from 66 to 108. He wishes to lie down, closes his eyes and lies in a stupor without response. Under repeated requests he gets up, stares in one direction, mumbles prayer words, and at one time looks at his hand and says, "There is the stigma of the Christ." No relevant communications can be obtained from him. There is definite catalepsia and cerea flexibilitas.

On May 9, 1933, at 8.55, patient is given 1.2 ccm. of adrenalin. The blood pressure remains constant at 143/55. There was no change in heart rate. His behavior, which had been rather withdrawn at the beginning, remained so throughout the interview.

On May 9, 1933, he was given cocaine. He became excited, restless, talkative, developed ankle clonus. He became quite self-assertive and suggested that he present his solution of the world problems.

Physiological Alterations Under the Influence of the Drugs.

In order to allow a survey of the different effects of our drugs on the variety of physiological functions to be tested, we placed all alterations observed together in one table for each drug.

The maximum change in leucocyte count, blood sugar level, reflex latency period, and chronaxia values of the arm muscle groups are stated in this table. This does not give a complete picture of the alteration. In some instances an initial increase was followed by a subsequent decrease. Nevertheless, we thought comparison would be best possible if the maximal deviation was recorded.

The small space which is available for this study does not permit more detailed accounts of all our findings. We shall, however, give examples of the complete curve of changes for a few patients afterwards.

The effects of adrenalin are shown in Table 1.

There is no significant change in behavior and experience. There is a marked increase in blood sugar level in all cases, a marked change in leucocyte level, increase in low counts, decrease in high counts. There is a very slight change in patellar tendon reflex time. The chronaxia ratio is not altered in any instance. The chronaxia level shows a marked increase in psychoneurotic individuals, a less marked increase in paranoid schizophrenics, and no influence in schizophrenics with affective deterioration.

The administration of sodium amytal is followed by slight changes in leucocyte count and blood sugar level. (Table 2.) The patellar tendon reflex time remains practically unchanged.

The chronaxia level is little altered. The chronaxia ratio does not change if it is normal as it is in psychoneurotic individuals; it is restored to normal or nearly normal ratio if the condition at the time of administration is abnormal (equalization or reversal) as was found in some of our schizophrenic individuals.

The effect of mescaline, as seen in Table 3, is quite marked in leucocyte level, less marked in blood sugar level. The patellar tendon reflex time shows little change in average deviation, but in psychoneurotic individuals a marked increase in variability from stimulation to stimulation can be noted. The chronaxia level is influenced but little. The chronaxia ratio is reduced or reversed if the initial ratio was abnormal. It shows little change under the influence of the drug if the ratio before administration was reduced already.

The effect of cocaine is very variable. (Table 4.) Leucocyte and blood sugar level change without consistent deviation. The effect on latency period and chronaxia level and ratio shows marked individual differences.

The effect of hashish is closely similar to that of mescaline. A reduction in blood sugar level is noted in most subjects. The chronaxia changes are almost identical with those following mescaline.

Comparing all tables with each other, and considering the initial value of each experiment, it is noticeable that the patellar tendon

TABLE 1.

ADRENALIN.

Diagnosis.	Psychological.	Blood sugar.	Leucocyte count.	Reflex time.	Chronaxia *	
					Forearm.	Upperarm.
Psychoneurosis						
E. L.....	No change.....	75–118	8800–13000	20.2–20.8	$\frac{53}{26}-\frac{116}{58}$	$\frac{48}{24}-\frac{118}{28}$
L. D.....	No change.....	105–150	10000–7000	20.5–20.6	$\frac{45}{22}-\frac{76}{38}$	$\frac{41}{20}-\frac{74}{37}$
S. S.....	No change.....	105–200	10100–8200	17.4–17.7	$\frac{79}{40}-\frac{71}{18}$	$\frac{45}{22}-\frac{40}{15}$
Schizophrenia						
M. H.....	No change.....	88–174	6500–10800	16.5–16.8	$\frac{64}{40}-\frac{64}{40}$	$\frac{14}{18}-\frac{14}{18}$
M. P.....	No change.....	87–137	6200–7600	.....	$\frac{39}{19}-\frac{40}{19}$	$\frac{39}{19}-\frac{39}{18}$
V. W.....	No change.....	86–152	5800–11300	.....	.....	$\frac{38}{26}-\frac{30}{24}$
M. S.....	No change.....	95–190	10000–8200	17.4–18.0	$\frac{90}{54}-\frac{53}{27}$	$\frac{53}{24}-\frac{24}{12}$
G. F.....	No change.....	80–107	5500–10500	.....	.....	$\frac{40}{23}-\frac{72}{30}$
M. E.....	No change.....	76–116	6500–15000	.....	$\frac{54}{28}-\frac{61}{29}$	$\frac{30}{14}-\frac{41}{18}$
E. H.....	No change.....	.....	.....	.....	$\frac{27}{35}-\frac{38}{28}$	$\frac{17}{25}-\frac{18}{13}$

* The chronaxia values are arranged so as to give indication of the ratio. The extensor value is given above, the flexor value below the line. The reflex time is expressed in thousandths of a second.

TABLE 2.

SODIUM AMYTAL.

* The chronaxia values are arranged so as to give indication of the ratio. The extensor value is given above, the flexor value below the line.

The reflex time is expressed in thousandths of a second.

TABLE 2.

SODIUM AMYTAL.

Diagnosis.	Psychological.	Blood sugar.	Leucocyte count.	Reflex time.	Chronaxia.	
					Forearm.	Upperarm.
Psychoneurosis						
E. L.....	Hilarious.....	68–70	3200–8800	19.7–20.3	$\frac{53}{27}-\frac{44}{23}$	$\frac{47}{23}-\frac{42}{21}$
L. D.....	Sedate. Dignified.....	85–80	9200–9800	20.5–19.9	$\frac{45}{22}-\frac{52}{17}$	$\frac{41}{20}-\frac{48}{14}$
S. S.....	Talkative. Gay.....	65–100	6700–9000	17.1–17.8	$\frac{79}{40}-\frac{71}{18}$	$\frac{45}{22}-\frac{40}{15}$
Schizophrenia						
M. H.....	Less self-absorption.....	89–90	5000–9800	17.4–18.0	$\frac{38}{39}-\frac{35}{19}$	$\frac{17}{23}-\frac{26}{20}$
M. P.....	Increased contact. Loss of hostility.....	91–98	9500–4400	20.5–20.3	$\frac{54}{38}-\frac{79}{44}$	$\frac{18}{24}-\frac{74}{23}$
V. W.....	Very little change.....	62–69	.....	.....	.....	$\frac{40}{26}-\frac{40}{32}$
M. S.....	Increased contact.....	65–80	9700–11200	18.5–19.9	$\frac{90}{54}-\frac{53}{27}$	$\frac{53}{24}-\frac{24}{12}$
G. F.....	Less self-absorption.....	88–106	.....	.....	$\frac{76}{35}-\frac{79}{39}$	$\frac{27}{26}-\frac{35}{15}$
M. E.....	Friendly contact.....	87–82	7200–4950	.....	$\frac{72}{54}-\frac{61}{40}$	$\frac{32}{26}-\frac{22}{15}$
E. H.....	Increased communication.....	.....	.....	.....	$\frac{31}{23}-\frac{28}{19}$	$\frac{22}{10}-\frac{16}{08}$

TABLE 3.

MESCALINE.

Diagnosis.	Psychological.	Blood sugar.	Leucocyte count.	Reflex time.	Chronaxia.	
					Forearm.	Upperarm.
Psychoneurosis						
E. L.....	Anxiety. Self-absorption.....	70–85	6100–11400	21.0–22.1	55 – 22 / 26 19	48 – 64 / 24 17
L. D.....	Uncommunicativeness. Distressed.....	75–80	7200–6200	20.8–20.2	25 – 14 / 22 11	40 – 08 / 20 17
S. S.....	Self-absorption. Anxious.....	90–85	.....	16.5–16.9	81 – 73 / 40 52	44 – 62 / 22 35
Schizophrenia						
M. H.....	Hallucinations. Placid self-absorption.....	75–66	5800–10200	17.4–17.2	39 – 39 / 17 38	17 – 19 / 19 24
M. P.....	Pseudocatatonic excitement.....	80–108	9800–7100	19.6–21.8	58 – 38 / 24 40	56 – 39 / 20 40
V. W.....	No change.....	82–87	8800–9200	.....	.....	42 – 34 / 27 14
M. S.....	Self-absorption.....	70–85	.....	18.7–18.0	90 – 41 / 54 58	56 – 40 / 27 38
G. F.....	Angry. Antagonistic.....	.....	.....	.....	46 – 52 / 40 63	23 – 20 / 31 31
M. E.....	Self-absorption.....	80–95	.....	.....	39 – 42 / 34 42	39 – 14 / 20 13
E. H.....	Catatonic.....	.....	.....	.....	39 – 17 / 33 27	24 – 14 / 12 31

TABLE 4.

COCAINE.

TABLE 4.

COCAINE.

Diagnosis.	Psychological.	Blood sugar.	Leucocyte count.	Reflex time.	Chronaxia.	
					Forearm.	Upperarm.
Psychoneurosis						
E. L.....	Restless. Gay.....	88–78	7200–5900	.....	$\frac{52}{26}-\frac{41}{23}$	$\frac{46}{24}-\frac{62}{30}$
L. D.....	Irritated. Talkative.....	85–95	8000–7400	20.0–20.1	$\frac{44}{22}-\frac{70}{28}$	$\frac{39}{20}-\frac{58}{24}$
S. S.....	Garrulous.....	90–110	6600–7200	17.0–17.1	.....	$\frac{45}{22}-\frac{39}{30}$
Schizophrenia						
M. H.....	Tense. Restless.....	68–80	7400–6700	.....	$\frac{63}{31}-\frac{40}{24}$	$\frac{11}{24}-\frac{22}{11}$
M. P.....	Noisy. Aggressive.....	86–116	5100–7300	.....	$\frac{70}{38}-\frac{80}{40}$	$\frac{40}{17}-\frac{73}{13}$
V. W.....	Unchanged.....	.....	7300–8400	.....	.....	$\frac{45}{24}-\frac{44}{24}$
M. S.....	Talkative. Hostile.....	95–105	8800–7200	18.3–18.1	.....	$\frac{40}{29}-\frac{42}{19}$
G. F.....	Irritable.....	81–75	9000–12000	.....	$\frac{39}{23}-\frac{26}{24}$	$\frac{32}{15}-\frac{17}{37}$
M. E.....	Impulsive affect discharges.....	78–82	9200–10200	.....	$\frac{40}{21}-\frac{59}{67}$	$\frac{22}{11}-\frac{40}{36}$
E. H.....	Restlessness.....	.....	.....	.....	$\frac{26}{31}-\frac{28}{40}$	$\frac{11}{11}-\frac{18}{20}$

latency period is quite constant, as is the chronaxia level and ratio, in psychoneurotic individuals; in schizophrenic patients there are marked variations.

The chronaxia level and ratio can be studied particularly well if we compare the changes produced by different drugs in one and the same individual.

Charts 1, 2, and 3 present in graphic form the changes in chronaxia values during the course of the experiments. Chart 1 is

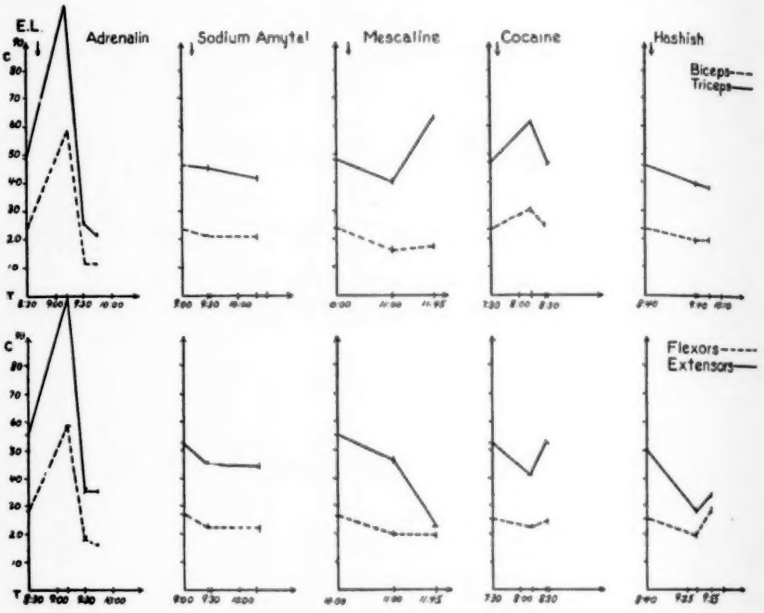

CHART 1.

characteristic for a psychoneurotic patient. One sees that in this picture the initial values are almost the same at each experimental session. The effect of adrenalin is a marked temporary rise in level without change in ratio. Sodium amytal produces little change in either level or ratio. Hashish and mescaline lead to a decrease in ratio without much alteration in the level of responsiveness.

Chart 2 shows in contrast the changes in a schizophrenic individual. Here we note a marked inconsistency of initial values. The effect of adrenalin on the level, which is so noticeable in nor-

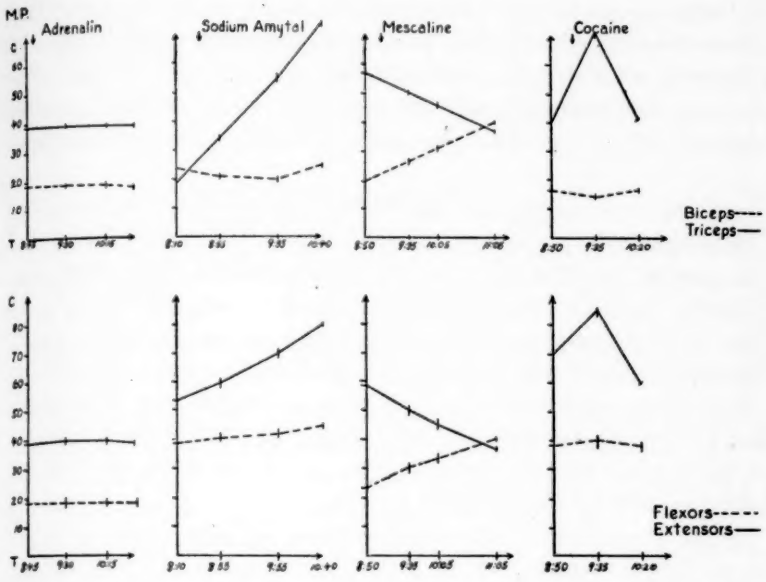

CHART 2.

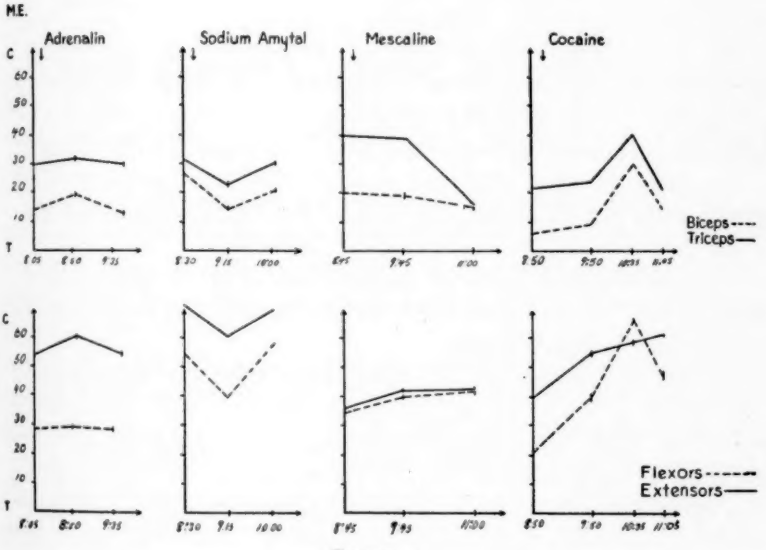

CHART 3.

mal and psychoneurotic individuals, is absent in this patient. The effect of amytal is noticeable in restoring the ratio which is missing before the administration; while mescaline shows no obvious influence on the abnormal ratio but reduces a fairly normal ratio to reversal. Chart 3 shows the same in a slightly different form.

Comments.

We have tried to observe the alterations of behavior which we can produce by adrenalin, sodium amytal, cocaine, mescaline, and hashish. We were able to record changes in experience and attitude which correspond closely to the changes previously reported: Increased contact and increased interest in the outside world resulting from sodium amytal and cocaine, withdrawal of interest and increasing absorption in inner experiences and their changes resulting from mescaline and hashish. Similar findings, meanwhile, were reported by Deschamp.[12]

In the present study we added a survey of alterations in several physiological reactions of the organism. In addition to the customary recording of the neurological status, blood pressure and pulse rate, we considered blood sugar level, white cell count, and as a neurophysiological indicator, the patellar tendon reflex time, and the chronaxia values of the right upper extremity.

All of the drugs produced changes in most of the physiological sectors of the organism which we studied. The effect of mescaline and hashish, although most obvious in the field of psychological behavior and cerebral functions, can be demonstrated in sugar metabolism and white cell count also; on the other hand, the effect of adrenalin can be shown not only in metabolic changes but also in the neurophysiological state of the individual.

The physiological effect of the different drugs is not uniform; it varies just as much with the reaction types of the individuals as does the psychological effect.

One set of our findings, the blood sugar level and the leucocyte level, is fairly typical for each drug, and changes little from person to person. They can serve, therefore, as evidence for the fact that the drug actually has taken effect in one sector of the organism. The spontaneous changes in blood sugar level and leucocyte level which we recorded were quite small when comparing the initial values of each experiment.

That adrenalin can influence the blood sugar level was known for some time. Cannon studied the relationship to emotional states and Bowman [7] discussed the relationship of blood sugar level to certain psychiatric conditions. Changes in leucocyte level resulting from adrenalin injection were recently reported by Martin.[21]

Similar effects of Cannabis Indica and their relation to the prevailing blood sugar level were reported last year by a co-worker of Beringer.[5] He found that Cannabis markedly reduces the blood sugar level. The physiological effects of amytal have been studied recently by a number of authors and were discussed by Solomon.[25] For our purpose it suffices to state that the positive effects on both the sugar level and leucocyte count can be demonstrated in almost every experiment. This effect may be marked in subjects where the effect of a drug, for instance of adrenalin, is not demonstrable by our neurophysiological indicators.

One of these indicators, the patellar tendon reflex time, shows limited changes under the influence of the different drugs. One finds increased variability as the main result of mescaline and cocaine in psychoneurotics which cannot be demonstrated in the schizophrenic group. We did not dare to use them for any generalizations until more experimental data are available. Our findings have relation to the results of Travis,[27] who was able to show in group studies a significant reduction in reflex time under the influence of alcohol and in relationship to certain types of mental disease.

By the second indicator, the chronaxia, we obtained more definite material. Chronaxia findings concerning this were obtained from our own previous work on the effect of our drugs on normal individuals and from the extensive studies of Altenburger [2] and Kroll concerning the effect of adrenalin on motor and sensory chronaxia. The following may be said for normal individuals: The spontaneous variations in normal individuals of chronaxia level and ratio are rather limited. Adrenalin produces a marked rise in motor chronaxia level without changing the ratio. Sodium amytal leads to a slight drop in level and no change in ratio in normal individuals. No findings for normal persons are available with reference to the other drugs used.

Our subjects comprise three cases of psychoneurotic adjustment, two patients with paranoid adjustment but with fair preservation of affective responses, and five patients with marked deterioration of affect and more or less readjustment in the form of schizophrenic defect conditions.

In our group of psychoneurotic adjustments, the level of responsiveness shows no *spontaneous* changes. It is markedly influenced by adrenalin and other drugs. The change in the state of excitation in the vegetative system seems to find expression in changes of the level of the neuromuscular responsiveness (comp. Hess,[14] Altenburger,[2] Achelis,[1] Stein.[26]) The normal ratio of 1 : 2 is well preserved under the influence of adrenalin, cocaine, and sodium amytal; mescaline and hashish, however, tend to lead to a reduction of the proper ratio, corresponding to the mental change in reaction pattern which they produce.

In the schizophrenic group the level of responsiveness shows spontaneous variations, as does the ratio. There are numerous instances of loss of ratio, reversal of ratio recorded at the beginning of the experimental sessions. Several French authors[9, 10] made similar observations. In the group of patients in which the psychopathological observation showed absence of affective responses, adrenalin produces no change in the chronaxia value, although the effect may clearly be seen in the rise of the blood sugar and leucocyte levels.

Sodium amytal, which has no marked effect on normal ratios, shows a marked effect in restoring the normal ratio in cases where it is lost or reversed at the beginning of the experiment. Mescaline and hashish both tend to produce equalization of the values, or reversals in case of initial normal ratio but show no effect if this process already has taken place. The power of mescaline and related drugs to produce states of absorption with abnormal motor conditions of catatonic-like nature was seen by De Jong[11] and Deschamp[12] before.

For us it is important to note that the drug effect becomes manifest only if the total pattern of the individual is adequate for it.

On the basis of these findings we venture the following suggestions for further psychopathological work: In psychopathology we are forced to develop certain concepts which, at the surface, seem to have little use in physiology.

We speak about different levels of regression, of withdrawal of interest, loss of effectualness in thinking, and other mental activities.

In the present study we found that we can demonstrate correlated processes in physiological patterns if we arrange an appropriate experimental situation.

Loss of proper chronaxia ratio was found before also in a number of organic conditions resulting in loss of normal relationship between the central nervous system and the peripheral organs. It also was found as the result of toxic conditions. Numerous observations of this sort are compiled by Stein.[22] Furthermore, in small children the normal level and ratio of motor responsiveness seems to become established only in the course of the development of several months. Reports to this effect are found in the studies of Banu [4] and Bourgigon.

In the light of such observations the following relationships seem worth mentioning on the basis of the present study in psychopathology: On the one hand, we have observed in certain patients withdrawal of interest from the outside world and regression to more primitive types of mental behavior from a previous level which was in closer coherence with environmental demands. On the other hand, we found a simultaneous transformation of neurophysiological patterns, resulting in loss of the normal level of responsiveness and of the normal pattern of coordination which was established as most suitable to meet the environmental demands placed upon the motor system. Here, the same as there, we have a regression to a more primitive, less adequate level of behavior. A drug which restores outside contact also restores the normal level of coordination (ratio). A drug which facilitates withdrawal from the outside also produces a regression in the pattern of neurophysiological responsiveness.

Whether a drug will produce a loss of ratio or not, depends upon the state of the individual to whom it is applied, and this variability of physiological reactions corresponds to the variability of psychological behavior.

Our material is not large enough to permit any inference of clinical value. Our equipment needs further development and theoretical problems have to be met with. If our findings should hold for larger groups, the combination of pharmacodynamic and

neurophysiological study may help to gain information about a patient's emotional state more rapidly and securely than our present methods of observation allow. Ultimately this method may help us to find a new common basis on which we can try to move ahead at least a few steps toward the goal of bridging the gap between psychopathology and physiopathology.

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AN EXAMINATION OF THE CLINICO-PATHOLOGICAL EVIDENCE OFFERED FOR THE CONCEPT OF DEMENTIA PRÆCOX AS A SPECIFIC DISEASE ENTITY.

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I. Historical Survey.

The formal introduction of the concept of dementia præcox, as a specific disease entity, occurred in the year 1898. There were many opponents to such a radical change of viewpoint; nevertheless the psychiatric world, as a whole, welcomed the new concept as a "new orientation, with a provocative challenge. . . . ." Yet, as Adolf Meyer has stated, it presented "no better understanding of the disorder, by calling it a metabolism disorder like myxedema and paresis, such totally different entities as we know them today. . . . ." There was, however, the "strange fact" of the appeal "to a nosological principle of cause, course, and outcome, where the cause was declared to be unknown, the description of the course flirted with the vesania pattern (the general or total insanity concept of Kahlbaum), and the outcome not any too certain, although in principle tending to be one of apathetic deterioration or at least a defect with apathy and oddities."[1]

It was this "appeal," this desire for a promised land where "a unity of cause, course and outcome" would hold full sway, which motivated great numbers of workers to investigate a heterogeneous group of cases, and brought to the front the concept of deterioration. The question naturally arises, how did this concept of dementia præcox as a specific disease entity begin to take shape, as the great avalanche of fact and fancy moved slowly forward with the years, its progress being continually urged onward by many

enthusiastic workers. In order to properly evaluate its present status, we must of necessity retrace the various phases of its development.

In 1809 Pinel[2] described the clinical picture of "Idiotisme or the Obliteration of the Intellectual and Emotional Faculties." "It is more or less the absolute abolition of the understanding and emotion," he stated. "The greater part of these idiots do not talk, or limit themselves to muttering inarticulate sounds. Their facies remain inanimate, their senses dulled, their movements automatic, a state of habitual stupor, a type of invincible inertia making up their disposition." Pinel also discriminated between false judgments which he termed *délire* (insanity with delusions) and that of *démence* (dementia), "in which there is no judgment true or false—the ideas exist as if they were isolated; . . . . they are in no wise associated—in other words, the thinking faculty is abolished."

The cause of such a grave disorder appeared to the author as pluralistic: "the abuse of stimulating pleasures, narcotic beverages, head traumatism, sudden fright, or marked grief, hard study without guiding principles, intracranial tumors, one or several apoplectic attacks, and excessive bleeding in the treatment of other types of general insanity (manie)" are held to be equally responsible etiological factors.

It was not until 1860 that Morel,[3] a countryman of Pinel, first used the term "démence précoce" to describe a condition in a boy of 14, "who had forgotten all that he had learned, his intellectual faculties, formerly brilliant, suffered a very disturbing period of arrest." Morel placed these patients in the third class of the "folies héréditaires" which were to him disorders of a degenerate heredity with which were associated the stigmata of degeneracy as seen in the shape of the head, short stature, and sterility. In commenting upon the intellectual defect, the author stated, "this diminution, which is a constant factor in dementia, is also the natural result of the age and the inherent decadence of human nature." The acumen of the clinician, however, did not overlook the psychological setting. He mentioned the patient's inferiority feelings in regard to his short stature, the hatred of his father, the gradual increase of uncommunicativeness and brooding with

tendencies to solitude—all these are mentioned and included as part of the "degeneracy" (p. 566).

Even before this preliminary attempt at the delimitation of the dementia præcox concept was undertaken by Morel, another disease entity which was to play such an important rôle in its later development already had been described. Bayle, in his doctor's thesis (Recherches sur les Maladies Mentales, 1822), for the first time, had made the correlation between "general and complete paralysis and disorders of the mind, when they developed side by side," and had ascribed the occurrence of both of these phenomena to a chronic arachnoiditis—"a distinct disease with a distinct pathological anatomy." [4]

II. Macroscopic Anatomical Descriptions and the Development of the Paradigm of General Paralysis.

Another link in the chain of conceptual weaving, which was soon to lead up to the formulation of a fixed disease entity, was forged when Hecker (1871), following the clinical outline laid down by Kahlbaum, first elaborated the symptomatology of hebephrenia.[5] He ascribed to Kahlbaum the differentiation of this disease concept from that of general paralysis, and attempted to establish well-defined clinical criteria for this newly created disease entity. "The disease, in the majority of cases, apparently follows a deeper emotional alteration, with the decided symptoms of melancholia, which finds its expression to begin with in an indefinite, nameless sadness and lowering of mood, and then gradually crystallizes into definite, although usually fleeting delusions." It was the peculiarity of this "melancholia" that was particularly stressed. "Often it has the appearance as if the patient were playful or coquetting. . . . . Alongside of a certain craving to lament over the terrible distress, the misfortune which had struck him, or over the sins which he had committed, the persecutions to which he was subjected, the patient often cannot suppress a tendency to laugh and tell silly jokes" (p. 402).

"The essential characteristics of hebephrenia are: its sudden onset in connection with puberty, the successive or alternating appearance of various clinical forms (melancholia, excitement, confusion), its extremely rapid termination into a mentally weak-

ened condition and the peculiar form of this terminal imbecility whose signs may be recognized in the first stages of the disease" (p. 419).

In regard to the etiology Hecker stated that "we deal chiefly with individuals who have been retarded in their somatic, and especially in their mental development, through known or unknown relationships to somatic disease, especially to frequent head trauma and onanism." Under the head of "pathological anatomy," Hecker presented *the one case* which had come to autopsy. He wisely reserved comment in this case, Julius C. (V), who entered the hospital at 19 years of age, and died of pulmonary tuberculosis at 25. The protocols read: "markedly injected pia, darkened cortex, brain pale on cross-section, moderately moist, somewhat enlarged ventricles, hyperæmic cerebellum."

As to prognosis, it is summed up in one word—"incurable." Nevertheless, Hecker includes one case report in which the patient recovered, and later was married, although he is in serious doubt as to the "complete" recovery of this patient in the absence of adequate catamnestic data. Already we began to appreciate that element of fatalism which was to accompany the concept of dementia præcox throughout the years until the present day—prognosis "incurable," and if recovery occurs, great doubt is to be expressed as to the original diagnosis, or as to the degree of "normality" which can be attained following such a "disease process." Hecker had sounded the criteria which were to leave a well-defined impression: (1) there is a definite clinical course in connection with puberty (ages 18-22), (2) the "disease process" rapidly reaches a characteristic imbecility (Blödsinn), (3) from which there is, as a rule, no recovery.

Kahlbaum (1874) added to the list of conditions ending in a terminal deterioration, what appeared to him to be another clinico-pathological entity—"die Katatonie oder der Spannungsirresein." "Katatonie," to him, was a "*disease of the brain* with a cyclic alternating course, in which the series of psychical symptoms—melancholia, mania, stupor, and confusion, and finally deterioration—appear . . . . and in which, alongside of the psychic symptoms, there exist processes with the general character of a spasm."[6]

This disease process represented a pathological entity to Kahlbaum (p. 82). ". . . . When we review the facts of the anatomical

findings in their entirety, we find that almost all the tissue elements of the intracranial organs show a common genetic process, whose development can be followed in cases of varying duration." The record of the specific findings reads as follows: (1) *recent cases* presenting brain tissue softening, and serous infiltration, exudate formation of the arachnoid, with larger deposits in the base; (2) *older cases* presenting a retraction of cerebral tissue, and the organization of soft ependymal exudates.[6]

It was to general paralysis that Kahlbaum turned for both a differentiation and a corroboration of his newly separated clinico-pathological entity; thus it is "general paralysis, which in the present state of pathological anatomy, can alone be considered as the basis of comparison. . . . ." And it is because of a lack of similarity of the findings in catatonia with those in general paralysis, that Kahlbaum insisted upon a special clinical group being set apart. In general, this investigator found that the same morbid processes "which were to be found in all mental diseases were present, but modified to some extent in catatonia." The first phase (hyperplasia) is "fleeting," and the second phase (atrophy) "appears late." The microscopic findings are reported as revealing nothing characteristic, but with the optimism which lies in the anticipation of an increase in the knowledge in regard to the normal cortex, the author expresses the hope that a more characteristic histopathology will be revealed in the future.

From a historical viewpoint Kahlbaum had succeeded in providing a substantial anatomical substrate for his disease concept, and had thus saved it from pathological disrepute. In his own words, "catatonia, as well as hebephrenia, ... can, therefore, still be protected from the submergence into the French concept of degeneracy which is unfortunately over-flooding the German literature" (p. 99). The direction of the "avalanche" of research had been set for some time to come. Thus in 1919 Renaudié[7] writes: "*Dementia præcox* is a dementia with organic lesions, and these lesions have zones and tissues of election." He cites the work of Kahlbaum as the first report of "a congestion with exudation of the encephalic vessels and the softening of the cerebral cortex" in this disease process. The "pathology" of catatonia had become the "pathology" of dementia præcox.

The English school, as represented by Maudsley (1895), discussed "the primary or acute dementias." Under this heading we find "the acute insanity befalling adolescents of fragile nervous constitution, especially those who have exhausted a weak nervous system by frequent self-abuse or other excesses . . . ." [8] This group falls into the larger category of the "pubescent or adolescent insanities" which are "badges of hereditary degeneracy" appearing at a period in which "blind longings and cravings, undefined aspirations, tremulous pantings for the unknown, large vague enthusiasms accompanied by a dreamy sadness" present themselves. "Ardent proposals to found a scientific classification of insanities on morbid pathology may for the present be ranked with not less fanciful proposals to define and describe in a nerve tract the thrill of love, the quiver of anger, the physical conditions of a warped thought or morbid suspicion." The "philosopher" having expressed himself, the "scientist" approaches the problem of the organic findings in the psychoses more warily, and with the traditional approach by way of comparison with general paralysis. In general "there are thickenings and degenerations of blood vessels and cortex." The morbid anatomy is epitomized by general paralysis, which "might be said to present the morbid changes of ordinary insanity in distinct and accentuated characters, so that they are more easily read, as it presents their symptoms in more dramatic form, all the inflammatory signs and products at first, followed in due succession by the degenerative and destructive effects." [8]

III. Fundamental Histopathological and Clinical Descriptions.

It was in 1896 that Kraepelin was led to think of dementia præcox as a "distinct disease, on the one hand, from the overpowering impression of the states of dementia quite similar to each other which developed from the most varied initial clinical symptoms; and, on the other hand, from the experience connected with the observations of Hecker that these peculiar dementias seemed to stand in near relation to the period of youth." [9]

The morbid anatomy (Chapter VIII, p. 213) [9] involves "a severe and widespread disease of the nerve tissue." Alzheimer is cited as describing "deep spreading changes in the cortical cells

especially in the deep layers, swollen nuclei, shrunken cells," and Nissl "invariably saw widespread cellular disease"; again, "deposits of lipoid products of decomposition" are reported. "All of these most severe morbid residue could be demonstrated in marked predominence in the second and third cortical layers." Sioli, Moriyasu, Wada, and Goldstein all report cellular changes, and Alzheimer adds his observations of the "amœboid hyperplasia of the neuroglia."

Kraepelin, the astute clinician, did not overlook the "psychic peculiarities" of his patients. He tried to evaluate "the shy, retiring dispositions who lived only for themselves" in terms of "constitutional injuries which lower the power of resistance to the cause of dementia præcox or whether it is a case of the first indications of the disease reaching back into childhood," and the weight of his opinion rested upon the latter theory. Kraepelin discounted infections, trauma, or acute intoxications as decisive factors but indicated the probable agent to be an "autointoxication." In the discussion of specificity or the source and kind of toxin there is only an apology for the "incomplete state of our knowledge."

We may epitomize his etiological viewpoint as follows: *"The fact is decisive that the morbid anatomy has disclosed not simple inadequacy of the nervous constitution but destructive morbid processes as the background of the clinical picture."* We have laid down here what appears at first to be an unavoidable conclusion from the formidable array of anatomical facts which had been gathered up to the year 1909.

It was chiefly upon the work of Alzheimer that Kraepelin had formulated his theories of dementia præcox as a destructive process of the cortex. Alzheimer first emphasized the significant part played by the ganglion cells in the general pathology of the psychotic processes in 1897.[10] The frequent occurrence of acute or chronic changes in the ganglion cells, and more frequently the falling out of groups of cells limited to one or another cell layer were stressed in this contribution. Alzheimer also pointed out at this time that injuries to these ganglion cells (whose end processes had been traced in the cerebral cortex by Golgi and Rámón y Cajal) may be assumed to be responsible for disturbance of cortical function.

From the onset we find an attempt made to correlate the findings in catatonic conditions with those of general paralysis. "There is

in catatonia, no doubt, a pathological glial fibrosis in the cortex limited to certain cortical areas." Again, "In general paralysis as in catatonia such pictures are frequently observed." These glial fibrous bands are generally described as surrounding the ganglion cells; frequent degeneration of the cell body or nucleus of the ganglion cell and regressive pigment changes in the body of the spider-like cell are reported. But Alzheimer also notes that the pigmentation of the cell body has been reported in epilepsy, arteriosclerosis and in senile dementia as well as in catatonic conditions.

The marked glial reactions are contrasted with the "passive responses" of the glia in febrile deliria, intoxications and exhaustion psychoses. "In intoxications, varying with the chronicity and acuteness of the process, there is a marked glial response which is characterized by nuclear segmentation with little pathological fibrosis." By contrast with deterioration insanity (Verblödungsirresein) one finds an *outspoken glial proliferation* with a pathological fibrosis limited to a definite portion of the cerebral cortex. The clinical entities of paranoia and melancholia are not correlated with any histological findings; in the words of Alzheimer, such histopathological observations are "still lacking." He finally concludes that "*the reports of pathological findings in the central nervous system, by themselves, cannot give rise to a definite decision when the clinical facts are not free from objection.*"

The concept of the disease entity, dementia præcox, gained another staunch adherent in the person of Diem. In 1903 he reported 19 cases, all of which he characterized as "simple dementia."[11] This investigator summed up his findings as follows: "Alongside of the clinical pictures of hebephrenia, catatonia, dementia paranoides, and the paranoid form, all of which end up in the characteristic deterioration of dementia præcox (after Kraepelin), there is still another clinical type which has a similar termination, with the same disturbance of intelligence and emotion, but here, the beginning is uniformly simple, insidious, ... without acute exacerbation and remissions, ... without sense disturbances and delusions, and without the characteristic phenomena of catalepsy, tics, mannerisms, stereopathies, negativisms, mutisms, etc." "After some years there appears a rather fixed condition. The clinical course is best introduced—like the analogy with the

simple form of progressive paralysis—as the simple demented form of dementia præcox, in general, dementia simplex."

Although in this paper Diem reported no autopsy findings or specific microscopic defects, he fixed the clinical concept of deterioration in the mind of the psychiatrist, by the introduction of still another clinical type which presumably had in common with the other forms of dementia præcox a similar termination—that of deterioration. The time was opportune for a counter-reaction. The original concept of "démence précoce" had been lost sight of in the lack of delimitation of the term, to include any and all disorders which terminate in the scrap heap of irreversible psychobiological bankruptcy.

IV. The Genetic-Dynamic Concepts of Adolph Meyer (1903-1910).

By 1910 Meyer's studies had begun to focus themselves upon "the factors which are apt to shape or undo a life—specific defects or disorders of balance with special tendencies and habitual ways of bungling and substitution and a special make-up which is liable to break down in specific manners." [12] This concept had its origin in an earlier attempt at an analysis of the neurotic constitution (1903). At that time he had emphasized the existence of a deterioration type alongside of the psychasthenic, neurasthenic, hypochondriacal, hysterical, epileptic, unresistive, maniacal-depressive, and paranoic types. "In cases of dementia præcox," he wrote, "we find over and over an account of frequently perfectly exemplary childhood but a gradual change in the period of emancipation." But upon closer investigation, Meyer demonstrated that this exemplary conduct is found to be "an example of goodness and meekness rather than of strength and of determination, with a tendency to keep to the good in order to avoid fights and struggles." [13]

Later on we find "vivid religious interests, whims, irritability, moralizing, sensitiveness to allusions of pleasures driving the patient into seclusion." Again, "headaches, hypochondriacal complaints, day-dreaming, utterly immature philosophizing," and above all, "loss of directive energy and initiative without obvious cause such as well-founded preoccupations, except the inefficient applica-

tion to actuality." All of the traits may be of transient duration but the author emphasizes the fact that they are not the symptoms of "neurasthenia," but "the beginning of a deterioration, more and more marked by indifference in the emotional life and ambitions, and a peculiar fragmentary type of attention, with all the transitions to the apathetic stage of terminal dementia." [13]

In 1910 Adolph Meyer summed up his genetic-dynamic viewpoint as follows: "The picture as a whole makes the diagnosis. There are no decisively pathognomonic facts. The deterioration gives the disorder its name; but it is not always realized; the essence of the process is a hypothetical toxic influence—entirely hypothetical—with definite brain lesions, also vague and not explained." His interpretation of the organic findings went a step further. "They (the brain lesions) are very different from those of general paralysis in their nature, and as to autonomy or origin. They are most akin to fatty involution of the brain tissues, probably incidental to the disorder of function as is the brown atrophy of the heart and fatty degeneration of muscles or liver. . . . . The occasional late recoveries of apparently demented patients and the peculiar clearing up of some cases with intercurrent disease makes one doubtful about the 'profound deterioration of the cortex.' . . . ." [12]

Here we find a new aspect to the problem but unfortunately one which was buried under the avalanche of specific brain findings. Nevertheless, Meyer's warning that there was something valuable to be gained by working with total settings and the facts at hand as contrasted to the precarious protection gained in working under cover of "fatalistic and analyzed conceptions of heredity, degeneracy, and mysterious brain diseases" had made a distinct impression on many independent investigators. His words at that time set a definite goal for future research which many investigators had been seeking: "I am convinced that the complete account of the cases in their concrete settings is bound to be the best and only safe basis for deduction—realizing that I left untouched many legitimate queries especially those about the non-mental or so-called physical components of the reactions, and the possibility of accounting for them with habitual substitutions and habit conflicts." [12]

V. The New Impetus Given to "Specific Findings."

The publication of the findings in 55 cases of dementia præcox in 1913 (of which 13 were chosen as uncomplicated) by Alzheimer [15] was the starting point of the more recent emphasis which has been placed upon histopathological specificity. "Amœboid glia" were found in six "acute" cases. The alteration found in these brains is one in which "the ganglion cells of one layer may be oriented with their apical processes in various directions, a condition which does not occur normally. The changes are most pronounced in the *second and third layers;* where the changes are not so pronounced there are found numerous ganglion cells of the upper layers in a state of *sclerosis, a condition frequently found also in cases of paralysis in the same region.* This sclerosis is practically always associated with a lipoid degeneration, the degree of which varies in different cases and is not necessarily most pronounced in the individual who suffered longest from the affection, but may be extremely pronounced in individuals in whom the affection lasted from two to three years. It is possible that the lipoid substances later disappear from the cells." . . . . "On the basis of our findings we may state that the injuries of the elements of the cerebral cortex are quite considerable in dementia præcox. In some of these cases the changes are so pronounced that a pathologico-anatomical examination may furnish a differentiation of the condition from manic-depressive psychoses. However, conclusions as to the localization of the changes with reference to the clinical form of dementia præcox can only be drawn after our knowledge of tissue changes and concerning the physiological significance of the individual cortical layers is widened." [15]

From this time on we have the emphasis flatly placed on specific cortical layer injury in a condition tacitly assumed to be a clinical entity.

The investigations of Southard (1914) [15, 16, 17] brought the claims of specificity into sharp focus. The creator of the "brain spot and mind twist" hypothesis found the disease process to lie in those layers of recent evolution—"the supracortical and infracortical layers." His statements are straight to the point—in an analysis of five brains, four of them "looking in the general direction of dementia præcox, one of them manic-depressive . . . . *all* showed lesions in the supracortex, as well as in the infracortex, in the nature of

cell loss and of more or less disproportionate gliosis." The one case in which such lesions are absent, he designates as the brain of a paranoic, which presents " a hypersynthesis as opposed to a mental dissociation " (p. 18).[15] The latter alone is to be represented as a supracortical neuron destruction. For hypersynthesis (paranoia) the author has no explanation—" false reasoning without loss of power of sensory intake and power, without loss of memory images, without loss of power of expression, without any evidence of coarse loss of function, remains as a process not readily statable." [15]

In the case of catatonia, Southard feels that he is on more secure ground—" such manifestations are due to lesions (gliosis) in the infracortical layers." But the pathologist can make more conclusive statements than the foregoing; the correlations of several studies are summed up in his paper on the topographical distribution of these cortical lesions[17]: (1) " Delusions are, as a rule, based on frontal lobe disease, (2) catatonic symptoms are, as a rule, based on parietal lobe disease, (3) auditory hallucinosis is, as a rule, based on temporal lobe disease " (p. 664).

The fact that these frontal lobe lesions are absent in some of his cases with delusions does not cause this investigator to modify his statements in regard to the correlation of the cortical lesions and the functional disturbances. On the contrary, he finds it plausible to maintain that " these non-frontal cases of delusions are cases of hyperphantasia—an elaboration of phantasies not more than half believed, at any rate, not fully believed by the patient." It is the parietal lobe which may be " implicated in the function of imagination in these cases," and parietal disease is, therefore, responsible for " over-imagination."

The correlation between functional disturbance and the localization of organic brain disease is made a complete one in no uncertain language: (p. 188) " One can find a correlation between almost any symptom and some properly situated lesion in this case (2). The patient was deluded, hallucinated and catatonic, and had frontal, temporal and parietal lesions." [15] In case 3 (p. 189) " it *appeared* that she had auditory hallucinations which she tried to keep out by covering her head with her arms." (The content of these " hallucinations " was apparently not obtained at any time.) Again (Case 3), " an attitude of apprehensiveness, an early phase in which she constantly repeated the words ' Father Patterson,' led us to the

idea that she had delusions of persecution. . . . . The most pronounced regions of cell loss were in the infracortical portions of the parietal and temporal regions." Here we are presented with auditory hallucinations which are never reported and delusions of persecution which are never elaborated; yet these are the "facts" upon which are based anatomico-functional relationships.

Case 4 (of this series of five carefully chosen brains) was diagnosed as a manic-depressive. The patient was described as "extremely joyous, screaming and singing," who later quieted down and "agreed that she might have been a little insane." She was aware of her physical condition (active tuberculosis) and no memory defect was found. The diagnosis of this case was changed to dementia præcox because of the presence of "auditory hallucinations." "It is a moot point whether an auditory hallucinosis is found in manic-depressive psychosis." [16] We can do no better than refer to the investigations of Bowman and Raymond [18] on this "moot point." These investigators report that "for the manic-depressive group both males and females show more cases of having persecutory delusions and auditory hallucinations, and persecutory delusions and visual hallucinations than would be expected statistically." The evidence as presented in the papers under discussion [15, 16, 17] is inadequate when subjected to clinical re-examination. Yet, upon these five cases rest definite conclusions in regard to the relationship of focal lesions in dementia præcox and their functional significance.

The organic theories of dementia præcox gained many adherents during the vogue in which the theories of focal infection were heralded as being totally responsible for practically all of the ailments of man. Cotton [19] made a determined stand on this question when he stated in 1922 that "no one would delude himself that mental treatment of any character has been unsuccessful when applied to the so-called functional group (of psychoses). . . . . We are of the opinion that we can produce sufficient evidence both pathological and clinical to substantiate our opinion that the so-called functional psychoses are in reality disorders of the brain, and that the mental symptoms are caused by these disorders. Furthermore, ... function is dependent on structure. In other words, we cannot conceive of a distorted mind with a perfectly normal brain, a conception which unfortunately still has many

adherents. . . . ." The psychosis should be considered " a symptom, and often a terminal symptom of a long continued masked infection, the toxæmia of which acts directly on the brain." [19]

As to the source of these " continued " infections, the teeth are of primary importance. " Without exception, the functional psychotic patients all have infected teeth "; the tonsils, are also the foci of secondary infections involving the entire gastro-intestinal tract, " from constantly swallowing the bacteria originating in the mouth." The gastric infection is readily recognized by this investigator due to the presence of numerous types of bacteria associated with a low or absent content of hydrochloric acid. The author is enthusiastic as to the results of detoxication. First, all infected, impacted and unerupted teeth must come out; then the tonsils are removed; vaccines are used to combat the gastric infection. Cervical infections are treated by cervical enucleation or by hysterectomy, if the infection is of a diffuse nature. Excision of the seminal vesicles is recommended in cases where they are involved. (This infection is present in 50 per cent of Cotton's cases.) The " whole colon to the lower sigmoid is removed " in the presence of focal infection. Endocrine therapy has yielded poor results in the hands of this investigator.[19]

Several comments may be made concerning these enthusiastic statements and heroic procedures. There have been several investigators who have not been able to duplicate either the results or the bacteriological findings. Kopeloff and Cheny [21] state that the " removal of infected teeth and tonsils from 27 cases . . . . has been followed by no more mental benefit than was shown by a comparable group of 33 patients used as controls." The same authors report that " the bacteria found in stomach contents is not to be relied upon as a means for determining gastric infection." Goodall takes a definite position from a clinical laboratory viewpoint.[22] " The examination of the blood for organisms in the acute and recent psychoses by the usual ærobic bacteriological methods gives negative results." Also, " there is no sufficient evidence of differences, quantitative or qualitative, in the blood counts in the different forms of dementia præcox." Despite negative reports by other investigators Cotton is " firmly convinced that even if we did not have the evidence of cortical lesions in the " functional " psychoses, we would have to assume their existence if we accept modern

biological teachings." [20] Here we apparently have the crux of the problem—we must have a postmortem pathology or there can be no pathology, according to "modern biological teaching." Nevertheless, it was Alzheimer [10] who maintained that he could find no pathology (post-mortem) in melancholia and Southard [15] agreed that he was not cognizant of any histopathological changes in those cases with systematized delusions (paranoics).

This is not the place to discuss in detail the contention that if "we did not have the evidence of cortical lesions in the functional psychoses, we would have to assume their existence. . . . ." Previous conceptions of disease, demonic, theistic, humoral and magnetic have had their day and have given way in turn to what we are always pleased to call "the scientific approach." Nevertheless, all of these conceptions have led to fruitful therapeutic results. Rivers remarks "there can be no question that such processes (demonological) as I have recorded here are efficacious. Men who have offended one whom they believe to have magical powers sicken, and even die, as the direct result of the belief" (Rivers cited by Hart) [23] (p. 7).

The concept of integration (Adolf Meyer) as applied to psychiatry, makes a distinct contribution in its well-organized attempt to deal with these problems. It assumes that the disorders of man may be considered as "experiments of nature," and that the natural history type of approach which is common to all scientific inquiry is best suited to deal with these disorders. It attempts to handle sets of facts in the particular settings in which they occur, and therefore would speak of brain disorders in terms of demonstrable cellular change, always keeping in mind that the problem of person or personality-function remains to be dealt with in its own terms and settings. This concept emphasizes the individual person acting as a unit with a specialized type of behavior, the psychobiological, in which sign-function (pictorial, verbal, conceptual symbolization) is involved, and it speaks of structural, physiological as well as psychobiological levels at which disorders may arise. In handling human problems this concept employs as its "elements" the events as they arise in their individual biographic settings, as a direct contrast to the elements of the psychologist (ideas, images, emotions, etc.).

With such a working hypothesis we need not look for a "body" *or* a "mind," nor is it necessary to look for all "pathology" in a pathology of "brain" which is to be sought only at the post-mortem table; but to conceive of pathology as an experimental science whose business it is to inquire "what is wrong and why; to ask under what conditions these facts have arisen, what factors enter, and how can they be modified?" Such a concept is interested in function-inferiorities as well as organ inferiorities, and in function misuses as well as peculiar uses of function. Such an approach does not "assume" the existence of cortical lesions in order to have a "pathology." If such lesions can be demonstrated satisfactorily, well and good, but there are other facts which must be dealt with no matter what the character of the lesions may be that will be ultimately considered or rejected as specific for this type of reaction.

After the interpolation of these few words in defense of the utilization of psychobiological concepts as contrasted to a forcing of everything human into a concept of "lesion," we may proceed without more than mentioning the gigantic strides made in the understanding of the functional paralyses and personality disorders since the investigations of Janet, Adolf Meyer, and the more recent work of Morton Prince and Freud.

Sir Frederick Mott summed up his work in dementia præcox [24] in 1922 and concluded "that it is better to speak of primary dementia which may occur in the pre-adolescent period, adolescence, or post-adolescence; moreover, I am of the opinion that all the psychoses belong to one group and are genetic in origin." In cases of recovery "we may assume a suspension of neuronic function in the highest psychic level; but all these conditions I have found may end in a terminal dementia, in which the changes in the reproductive organs and in the brain do not differ from those met in the primary dementia of adolescence."

The facts upon which the above statements are based are illustrated by a "typical" case of "acute dementia præcox."

A young man of 19, of "average intelligence" and a "good athlete," with a direct heavy family hereditary loading (father died in an asylum; paternal aunt insane; maternal grandfather insane; one brother died of a bullet wound of the head; one sister admitted to an asylum for the fourth time). The illness began during his studies for an examination which he

"passed . . . . but became depressed when his sister went to the asylum . . . . had delusions of having committed a great sin, and asked for poison." The certificate stated that the patient was "*acutely melancholic.*" Two weeks after admission a "fading erysipelas of the left side of the face" was present. This patient died following an acute febrile illness (temp. 100°—102° F.) which was associated with a tachycardia (140/m), general hyperæsthesia, and sphincter incontinence.

The post-mortem examination revealed "very marked congestion and redness of the brain with minute hemorrhages especially in the medulla"; the testes weighed 8 grams each (10.5—14 grams—average weight). It is a most remarkable coincidence that the testicles of the brother (who died of the bullet wound) weighed the same. His testicles had been sent to the investigator as "*normal material.*" Yet in both of these cases "the nuclei of the spermatocytes show no mitosis." There are "relatively few spermatids," and "distorted spermatozoa" are present in the testicles of both brothers. "In fact, the histological cases correspond with those seen in cases of *advanced dementia præcox.*" The brain revealed "nuclear swelling, lipoid granules replacing Nissl granules, glia proliferation "especially marked in the supragranular layer of the pyramids"; . . . . similar changes (though less marked) were observed in the basal ganglia pons and medulla.

If we are to consider the foregoing case as one of "advanced dementia præcox," we must set aside what at first sight appears to be a predominating and motivating pattern of affect without outstanding incongruity in any particular sphere of behavior. The fact that this case was considered to be sufficiently "typical" to be published in detail in the memorial volume dedicated to Rámón y Cayal [25] and yet may cause others who review the same material to disagree as to the proper diagnosis, brings to the very front the need of setting up clinical criteria, which would provide a satisfactory delimitation of our diagnostic labels. What are the outlines which are available at the present time?

VI. Brief Outline of a Practical Delimitation of the Concept of Schizophrenia.

Adolf Meyer has described "a group of phenomena in the making or established, having in common incongruous fancies and experiences and reactions marked by more or less automatic fixed tendencies with a striking lack of plasticity, and in the main foreign to normal adult waking life. Either out and out odd or elaborate and dramatic akin to the primitive, hardly experienced or enacted in the present day except in hypnosis-like states or in states of religious

fascination, or in superstitions such as those of ' spells ' and peculiar influences.". . . ."All this develops either slowly or abruptly without any evidence of a leading or adequate disturbing affect or of the presence of flight of ideas or delirium; often in perfect clearness, on a foundation of an introverted brooding and fanci-shut-in tendency, generally on a basis of autoerotism or of more or less immature sex tension, and most far-reaching substitutive reactions allowing autistic tendencies or uncritical fancy to prevail without any adult critique with an acceptance of possibility as sufficient evidence of reality." (Unpublished material on the parergastic reaction set, used for teaching purposes at the Johns Hopkins Medical School.)

Here we find not only a conglomeration of symptoms fitting into a well-defined symptom complex, but a development out of a specific constitutional endowment into a specific mode of reaction. *It is a reaction set—not a disease entity.* The emphasis is placed *on the entire setting—where and when all this arises as well as in what particular manner and with what symptomatological coloring.*

According to Campbell [26] " the schizophrenic type of reaction seems to be characterized by diminished interest in and adaptation to the work-a-day-world, increased interest in subjective creations or fantasies which are emancipated from the control of ordinary logical or scientific thought, the frequent occurrence of hallucinations, odd and fragmentary behavior and utterances of little adaptive value in relation to the real present situation, representing fragments of modes of thought and behavior intelligible in the setting of the child or of primitive man." In regard to the presence of any disease process Campbell states that " we may accept the statement that the psychosis and the special personality are of the same cloth, without assuming that the personality is an early indication of a disease process which becomes more explicit in the psychosis." Again, " the psychosis can be looked upon merely as an extreme degree of the difficulty of adaptation of a person with a certain make-up confronted with the major issues of life . . . ." (p. 23). It is a dynamic concept that is presented—with every consideration for the fact that " in the infinite variety of human nature now one, now another, of the main characteristics of the schizophrenic reaction may be the more prominent, largely dependent upon variations in the individual endowment " (p. 26).

A very concise and practical application of the above criteria comes to us in the investigations of Greenacre who has reviewed the content of the schizophrenic characteristics occurring in affective disorders.[27] In a series of 40 cases she has found that "these symptoms fall according to their form into five classes: (1) distortions and misinterpretations of actual occurrences (delusions of reference and persecution); (2) influence and passivity feelings as expressed in automatism, mind-reading, electrical influence and similar phenomena; (3) hallucinations (particularly when the content is "vague, odd and incongruous"); (4) gross distortions of body sense and body appreciations; (5) incongruous behavior, occurring either episodically as "antics," or more persistently, but not in keeping with or apparently motivated by the prevailing affect.

The clinical criteria which have been outlined above will suffice for all practical purposes. We cannot but appreciate that much more can be said concerning the delimitation of the terms, dementia præcox and schizophrenia. May[82] offers many valuable suggestions in the way of a clinical outline. Willmanns by 1922 had collected 250 titles on dementia præcox; yet the field is not to be exhausted by these papers. For our special purposes we have laid down certain fundamental rules to which we may return with the discussion of new case material.

VII. Early Doubts and More "Specific" Findings.

Among the earlier attempts to critically summarize the status of the histopathology of dementia præcox we mention the protest of Hassin. This author readily acknowleged Brodmann's statement that up to this time (1918) all attempts made to define the possible functions of the layers of the cortex were nothing but "harmful speculations." He emphasized the "fact" "which seems well established that the ganglion cells of the second and third cortical layers, especially of the frontal lobe, are frequently found damaged in dementia præcox."

On the other hand, Hassin also asserted that these changes are "not uniform, typical or specific for dementia præcox since similar changes are to be found, sometimes even more pronounced, in any other mental disease or organic brain lesion" (meningoencephalitis, dementia paralytica, manic-depressive insanity, etc.).

"The disease cannot be diagnosed microscopically like dementia paralytica. . . . . This problem is practically in the same state as it was 20 years ago when Alzheimer first revealed his findings." [28]

Buscaino (1922), is more specific in his presentation of the intrinsic pathological anatomy. The areas of disintegration which this worker has described are characterized by the presence of "an abnormal substance, related by staining property to the substance X found in older areas of distintegration." Buscaino correlates catatonic manifestations with lesions in the corpus striatum (especially in the globus pallidus). He also reports frequent lesions of the dentate nucleus of the cerebellum and states that "it is not improbable that cataleptic phenomena are due to cerebellar lesions." More significantly the same author finds that the areas of disintegration are multiple, and found "first, in the *white substance* of the brain, then in the basal ganglia, and the underlying grey matter (particularly in the thalamus) *rarely in the grey substance of the cerebellar or cerebral cortex.* The résumé of the case histories in this series is very concise and gives one little to compare with the criteria we have outlined above. Thus:

CASE—.Martelli. Catatonic dementia præcox. Female. Duration of illness, three years, eight months. Auditory and olfactory hallucinations; apathy; laughing and crying spells; sitophobia; catatonic with stereotyped movements such as rubbing the hands. Reflexes (patellar exaggerated). Died, aged 27 years. Tuberculosis (advanced) of entire left lung." [29]

The areas of disintegration were most rare in the cerebellar cortex, numerous in the pons, particularly in the anterior portion, and very few in the bulb and along the base of the fourth ventricle.

Reference to Table 1 of this paper [29] demonstrates that the contention of the author is well corroborated inasmuch as the *white substance* of F_2, the pons (zona anterior), the posterior brachium of the internal capsule, and the nucleus dentatus of the cerebellum are markedly involved (++++).

In the reports of Buscaino we no longer find any reference to the observations that "delusions are, as a rule, based on frontal lobe disease, and that catatonic symptoms are, as a rule, based on parietal lobe disease." There is, however, mention of some degree of involvement (+) of T 1 in this case where the investigator observed infrequent acoustic hallucinations, and we must remain satisfied with finding that modicum of agreement between different

investigators, who are attempting an anatomico-functional correlation in what at best is a heterogeneous group of reaction sets.

The trend toward more critical thinking in the pathology of dementia præcox received a greatly needed impetus by the investigations of Dunlap.[30] In 1924 he published his "preliminary observations on brains from carefully selected cases and a consideration of certain sources of error." He found that the "agreement is pretty general that there are no gross characters of special pathological significance in dementia præcox. . . . . There has been no verifications of Southard's claims that there are spots of greater resistance to touch in the cerebral hemispheres, and beneath these spots an increase in neuroglia." In regard to the dropping out of nerve cells in appreciable members (as claimed by Alzheimer, Nissl, Mott, Vogt, and others) the author finds no corroboration; "neither has there been found an increase of neuroglia masking atrophy."

Dunlap reported the findings in eight carefully selected brains (out of a possible 31 cases). These brains are collected under conditions which were as ideal as possible—(1) the patients were under 40 years of age; (2) the autopsies were performed almost immediately; (3) the cases were those which followed sudden death; (4) they were free of any doubts in the clinical diagnosis. In addition there were five "control" cases of similar age, whose autopsy material was collected under similar circumstances. These control cases had died suddenly of acute processes or accidents.

The following are the conclusions to which Dunlap arrives after working under these "ideal" circumstances: (1) That nobody knows what changes may occur in the nerve cells of normal persons as a result of processes going on in the body just before or just after death—processes in no way connected with any psychosis; (2) many of the so-called classical findings of other investigators were found in the normal controls (*i. e.*, the dark staining "sclerotic" nerve cells; cells with distorted nucleoli; destroyed cytoplasm, and the acidophile nucleoli (Mott); the excessive lipoid content of nerve cells (Cotton); (3) neither was there found an increase of the neuroglia, nor a loss of nerve cells in the outer cortical layers (Alzheimer) when a method of cell counting and photography was utilized in contrast to the descriptive method used by other investigators. (The control cases show almost "identical average" cell counts per microscopic field.) Dunlap con-

cludes his paper with a plea " for reasonable consistency in findings especially if far-reaching conclusions are to be based on them, and if we are to list dementia præcox among the organic brain diseases we must find at least in a fair percentage of cases some kind of pathology that will hang together."

In contrast to these " negative " findings and positive constructive suggestions for further research, another investigator at about the same time has published what he apparently considers to be the specific pathology of dementia præcox. Josephy working in the laboratory of Jakob begins his paper [31] with the statement that he is fully aware of the difficulties involved in obtaining worthwhile material. This investigator faces the problem squarely when he states that " there is no uniformity to the concept of dementia præcox," and feels that when one speaks of the histopathology of dementia præcox " one already takes for granted what remains to be proven—namely, the unity of the entire concept . . . . it is very apparent that dementia præcox includes a group of diseases, somewhat analogous to the group of organic diseases which fall into the paralytic, senile forms, etc." Because of the dependence on, and the confusion of clinical contributions, Josephy suggests that brains be examined independently of clinical descriptions, and that the results be grouped regardless of clinical findings. Here we have a distinctive step forward in the pathology of the psychoses—away from traditional labelling and pigeon-holing—a proceeding which Josephy states has led to the differentiation of the entire field of luetic brain diseases. This worker is impressed with the general lack of critical evaluation of data—" authors record brain changes in dementia præcox without mentioning or evaluating the terminal intercurrent disease or the age." Again, " single or too few cases are used in evaluating the histopathology of such a difficult problem."

Josephy's own contribution consists of an analysis of the material offered by 50 brains, and five " tubercular " brains of presumably " mentally healthy " persons to be used as controls. There are reported (a) changes in two " uncomplicated " cases both dying accidentally, (b) changes in those dying of chronic diseases, (c) changes in the basal ganglia and deeper brain tissues.

Essentially what we have contrasted in the two " uncomplicated " cases described by Josephy is the deteriorating process side by side

with a condition in which the affect plays the dominating rôle. The pathologist has the same answer for both of these cases—"an exfoliation of cortical nerve cells in the third and fifth layer, especially in the frontal lobe, to a lesser degree in the parietal and temporal lobes, and least, but to a definitely recognizable degree in the occipital lobe."

The heterogeneity of the case material is demonstrated by Case 6 which is termed a "possible late catatonia."

The patient is a man of 48 who developed a thrombosis of the left leg after an operation which followed an empyema. During his convalescence there were hallucinatory experiences and delusions (he heard voices, particularly that of his wife and stated his leg was to be amputated) in a setting of agitation and marked depression with suicidal brooding. He died suddenly following a pulmonary embolus. The mental health of this patient is described as being satisfactory previous to this illness.

The cause of death in the second group (chronic cases of dementia præcox) includes "rapidly advancing" tuberculosis, influenzal pneumonia, cystitis and cachexia, pulmonary embolus, and sudden collapse (cardiac?). As the influence of agonal states will be discussed later, no comment will be made at present concerning this significant topic.

Josephy's statements in reference to the "specificity" of his findings lead us to believe that he may be of the opinion that the findings are specific only for dementia præcox.

"First, the third layer is particularly involved; second, the falling out of cells is independent of the vessels (and of local disease processes); and third, the presence of intrinsic reactions of the glia is lacking . . . . If one finds these three characteristics, one should, in my opinion, ascribe the case with great probability to the deterioration psychoses, and one also may make a diagnosis on the basis of these findings" (p. 439). In summary, "the process is to be characterized anatomically as purely parenchymatous without any inflammatory components. . . . . Glia proliferation only plays a rare part" (p. 472).

Negatively, Josephy adds that he can find no changes in the choroid plexus (as claimed by Von Monakow). He considers the lipoid deposits of Buscaino as artefacts rather than true anatomic substrata of a disease process. Neither does this investigator report any significant changes in the endocrine glands, although he

comments on the positive findings in the testicles and ovaries reported by Mott; he adds: "How to explain his findings, the factuality of which is not to be doubted, must be put aside for the time being. . . . . One can only state that they are contradictory to everything that is known to us up to the present, and what I can see in the microscope" (p. 474). To this careful observer (despite his anatomical correlations), "it appears, in any case, that in the investigation of the causes of dementia præcox we will continue to fumble about in the dark for some time to come."

Among the more recent and comprehensive reports may be mentioned the work of Füngeld. The author's investigations include 11 "uncomplicated" cases and 20 controls.

He reports that in general the *third and fifth layers* (and fields 8 and 10) are principally involved but the *second and third* layers are also affected although to a much less degree. The *glial reaction* occurs chiefly in the *sixth layer*. The author finds no significant changes in the striatum, thalamus, cerebellum (dentate nucleus), or medulla; neither does he report any definite involvement of the choroid plexus. The final conclusions are that in schizophrenia "we are dealing with a primary parenchymatous degeneration which attacks certain layers and the extent to which it affects various fields varies considerably from field to field and for each field. One can recognize scar, terminal conditions, arrested phases, and acute processes in adjoining cortical areas." Although the author has found several of these phenomena in cases of intoxications and among his controls he emphatically states that "such findings are never found in their entirety in any form of intoxication" (p. 67). Nevertheless, "with the methods which are available to-day the anatomical diagnosis of schizophrenia cannot be made with the same assurance as it is made in a case of general paralysis or syphilis of the brain or Alzheimer's disease" (p. 4).

In the above descriptions we find a careful division into the three more or less distinct phases of a progressive parenchymatous involvement, but we can find no differentiating process which distinguishes catatonic features from expansive phantastic phenomena. Such "findings" can only be the beginnings of a "pathology" which is attempting to explain "what is wrong and why."

Among those reports which concern themselves with the histopathological anatomy of the nervous system in schizophrenia, we have reviewed Bouman's contribution [32] with a great deal of interest. He is very skeptical as to the positive value of the findings in the field of the endocrines. "The number of completely negative contributions is sufficiently considerable to justify the concept that these alterations must have a secondary significance, and that they do not belong to the essential changes which are met with in schizophrenia" (p. 528). However, in respect to the "essential" findings he is in firm agreement with Josephy that "schizophrenia is an organic disease of the brain, which consists of a primary parenchymatous degeneration of the cerebral cortex." "There is a laminary cellular death (in which the glia play no active rôle), which most definitely occurs in the third cortical layer" (although the second and fifth layers are involved to a lesser degree). "The distribution of these lesions is diffuse, spotty, independent of the blood vessels and occurs without the presence of any inflammatory signs." There are less well-defined changes in the myeloarchitecture. These latter alterations consist of "damage to the fibre bundles of the internal granular layer, with less clear-cut changes in the supra and infragranular layers" (p. 528).

This author includes his specific findings in the three brains which have been carefully studied. (There are no clinical descriptions of the three cases reported in this paper.) In these brains "the frontal and temporal lobes were chiefly attacked by the disease process. The "typical process" was found to be most marked in the gyrus insularis posterior and anterior; next the fields (Brodmann) 31, 37, 41, 45, 47; less marked in the fields 9, 10, 23, 39, 38, 4, 7 and 40. The process was still less prominent in 6, 11, 22, 28 and the gyrus central post., and finally absent or scarcely present in the fields 19 and 17. The cellular disease itself consisted almost entirely in a sclerosis and lipoid change of the ganglion cells, particularly in the third layer. But there was a falling out of cells and degeneration to be found in the other layers . . . . in the following order—2, 5, 6 and 4" (p. 529).

Any concept which is to explain such a diffuse process, according to Bouman's interpretation, cannot suggest a "selective process, such as is included in Vogt's concepts of pathoclysis." Neither does Bouman believe what Alzheimer suggested, "that there is a

special type in which only layer 2 or 3 are chiefly affected, nor does there appear to exist such a type as Josephy saw in the layers 3 and 5". Rather "it appears that the cortex is involved in all of its layers, here more, there less. It appears to me, also, that there it is of little significance to speak of a system disease, as Vogt does, and from which Spielmeyer turns away with justice" (p. 530). Finally, careful cellular counts which this investigator has made in an effort to compare the thickness of the various cortical layers, have demonstrated no selective falling out of cells in particular cortical layers. All this evidence convinces Bouman that there can be no data which support any such hypothesis as that of "pathoclisis" (p. 531).

The correlation between the various clinical phenomena and his particular anatomical findings is very cautiously approached. The author feels that "a review of the existing literature contributes no light here." There are, however, certain trends which he has observed in the three brains which he has so carefully examined. In those cases where "a marked development of the process occurs in the temporal lobe (chiefly in the fields 41 and 42) there are many clearly defined auditory hallucinations (*phoneme*) occurring in the clinical course. I can't decide just how far the hebephrenic and the catatonic form coincide with a marked extension of the process into the frontal and prerolandic regions. . . . . Perhaps one should consider it likely that psychomotor disturbances, hyperkineses, flexibilitas cerea, etc., are tied up with anatomical changes in the striatum" (p. 533).

Here we have the fruits of more than 50 years of serious investigation (1874-1928); a survey in which hundreds of brains have been subjected to minute histopathological research by conscientious and thorough workers. Yet it offers us almost nothing when we come to apply the results of these long years of labor in the explanation of why certain clinical forms of behavior appear and disappear. If we stop to think that the function of pathology is just this—to explain why something went wrong and under what conditions, we must state that all of the above investigations including the last have failed to supply a satisfactory explanation of the character and relationship of the etiological factors which are to be held responsible for that condition in any patient which we designate as schizophrenic.

VIII. Recent Period of the Critical Evaluation of "Specific Findings."

Such a paucity of clinicopathological correlation expresses only the negative aspect of this great problem. In order to arrive at a more positive conclusion we must offer some explanation for the presence of these diffuse laminary findings "especially in the third layer." If we accept three stages in the developmental history of the histopathology of schizophrenia as offered by Bouman[32] (1874-1900), (1900-1921) and (1921-), we may suggest that the fourth stage in the development of this history should begin in 1930, after being heralded by the carefully controlled work of Dunlap in 1924.[30]

In that year Spielmeyer wrote: "The question is so difficult that with greater experience many investigators have discontinued work because for the present it appears insoluble to them, and I, too, have done this."[33] Three fundamental errors in the evaluation of this type of research are stressed by Spielmeyer: (1) The effect of terminal acute diseases, (2) the great variation of the fatty deposit in the glial cell which is found in normal cases, (3) the presence of "fresh necrobiotic linear areas" in various parts of the brain. These findings according to Spielmeyer are not to be looked upon as acute exacerbations of the disease process *per se,* but are to be considered as part of a mechanism found under many conditions (intoxications, infections, etc.). Upon this point there remains much to be said, and Spielmeyer's investigations upon the findings in circulatory disturbances and the psychoses[41] supply many details of the behavior of these special mechanisms.

"Certainly," he states "I know many cases in which our anatomical analyses revealed no deviation from the norm." Nevertheless, in "carefully chosen cases," Spielmeyer (as Bouman and Josephy) finds a "cellular loss in the third layer of the cortex, as well as in the deep layers, sometimes with an enormous fat accumulation. In the acute stages we find active destructive phenomena in nerve tissue with progressive regressive changes in the glia, and often abundant destructive products. I believe, therefore, on the basis of such findings that dementia præcox is an organic process. This Alzheimer taught 20 years ago and Nissl too stressed it. . . . ."

But although, "the organic basis for the malady is established .... they do not suffice to separate this process and to establish it as an entity. And so we have not as yet a pathological anatomy of dementia præcox. *For we find such indefinite cellular losses in entirely different organic psychoses.* I cannot follow the optimism of many investigators who evaluate this finding more highly. These findings only indicate the organic nature of schizophrenia, not, however, the anatomic diagnosis and the differential diagnosis which we need so very much" (p. 244).[33]

In a communication dated the same month as Spielmeyer's contribution Bleuler[34] is more enthusiastic in regard to the correlation between the functional disturbances and its organic counterpart. He states that "these psychic mechanisms, as they are known at the present time, do not explain the whole disease. They are only possible if there is a certain predisposition of the brain, and this disposition in schizophrenia seems to be a progressive disease." This disease process has primary signs (disorders of affectivity and association) and secondary signs (autisms, negativisms, stereopathies, mannerisms, catatonic features).

Schizophrenic somatic responses such as "raised or lowered temperature, albuminuria, metabolic disturbances, gnashing of teeth, *Flockenlesen,* fainting fits or cramps, not infrequently followed by temporary paresis of the limbs or the language, pupillary disturbances, greatly increased idiomuscular reactions, vasomotor disturbances, edema, somnolence, disturbances of the chemistry of the body, especially of liver functions, abnormal protein of the spinal fluid," are to be looked upon as fundamentally similar to other physiogenic conditions such as "intoxications, fever psychoses, epileptic absences, meningitis, and encephalitis." ....[34] (p. 206).

Here we have a gathering together of totally different sets of symptoms and findings belonging to every possible somatic system without any attempts being made toward a formulation which would enable us to discover the underlying cause of a specific set of symptoms in any particular case. It is in this type of generalization and collection of symptoms without reference to adequate grouping which provides the weakest link in this method of approach.

But Bleuler goes a step further, and states that "we also find in the autopsy histological alterations of the brain tissue, which show some uniformity .... chronic histological findings *always*

correspond with the clinical chronic picture, and acute changes with acute ones. . . . ." (Alzheimer [15] denied that any such correlation existed.) From this time on (1930) we do not meet with such "positive statements" as to the specificity of the anatomico-functional relationships; a more liberal viewpoint was soon to replace it.

Ewald (1930) strikes this note in his concept of the dementia præcox-schizophrenia problem.[35]

"We sympathize . . . . with Kleist's concept of dementia præcox as a hereditary-degenerative system disease in which the system concept remains *an entirely open question.* We ourselves look for this system in a phylogenetic and embryological-functional direction as yet, *rather than in strictly localized anatomical brain lesions.*" Again, "psychology helps us only to supply and to study reaction types, the coupling of symptoms and syndromes, which, of course, may occur with special frequency in this disease. As to our classification, however, we must strive to remain in the biological somatic domain. Whoever on the basis of a schizophrenic symptom complex diagnoses instantly and with certainty a schizophrenia in the sense of the disease dementia præcox is guilty of a decided transgression in another category—even though he be right in 90 per cent of the cases. I object, however, to the disease concept of schizophrenia so commonly deferred to recently as showing a purely psychological content, a psychologic experience or reaction type which entirely overlooks the biological background—the malignant process and defect nature of the disorder." Here again we have a clear-cut statement and emphasis upon the "hereditary-degenerative system disease concept," one with which Morel would have heartily agreed some 70 years ago (1860), but one which leaves the "system concept as an entirely open question."

Gradually, as one reviews these facts, an impression begins to develop, and slowly comes into clearer definition that no one hypothesis can include the multiplicity of facts which all of the above investigators have gathered together and placed at our disposal. Especially does this seem true when recent researches by Malamud and Miller (1931) state that the methods of "serology and biochemistry again show us that very little changes in permeability, in iron content, in basal metabolism, etc., have been found, and whether these could be looked upon as causes, effects, con-

comitants, or even purely coincidental, has never been proven. . . . . One wonders then just what the basis may be of the very definite statements made by men like Bleuler or Kleist with references to the organic nature of the disease. At the same time, we find that very careful investigations by experienced workers have shown a definite relationship to exist between the onset and course of the psychosis and certain factors in the life of the individual." [36]

This dynamic relationship is in direct contrast to the "story" which is obtained in cases of "progressive degeneration of the cortical grey matter" in which a diffuse loss of ganglion cells in the third cortical layer also occurs (Alpers [46]). A progressive dementia and marked motility disturbances are reported in these cases, *yet we can elicit no "story" in the sense of a live human narrative which contains those strivings and failures with which we can readily sympathize and begin to understand.* It is the growth from within which impresses us in all of these cases, and one that has no dramatic value because we are dealing with an impersonal process which is determined predominantly by readily demonstrable cortical lesions.

The temerity of these and other investigators in questioning the definite statements of men like Bleuler and Kleist began a new era in the investigations upon this most baffling problem in histopathology. While these facts were being made to fit into traditional cubby holes by some investigators, others, working recently on subjects of a totally independent nature, have produced findings which throw light on the entire dementia præcox-schizophrenia controversy.

X. Contributions from the Field of General Pathology on the Causes of Elective Laminary Involvement.

In the field of general pathology, from the recent investigations involving the acute disease of the brain due to the functional disturbance of the circulation, we get our first start toward another aspect of our general problem. De Vries (1931) reported several examples of "acute cases of cerebral disease in which multiple lesions of vascular origin, electively localized in the cortical grey matter, form the most interesting part, or even the entire pathological process." [39]

In regard to the localization of these foci to the third cortical layer, De Vries quotes from the observations of Vogt, that there

TABLE I.

Table of Contrasting Results.

Investigator.	Year.	Positive findings.		Negative findings.	Clinical correlations.
		Gross changes.	Microscopic changes.		
Kahlbaum [6]	1874	*Recent cases:* Brain tissue softening. Exudate formation of the arachnoid with larger deposits in the base. *Older cases:* Retraction of the cerebral tissues. Organization of soft ependymal exudates.	..... No characteristic findings are reported.	.....	Positive findings correlated with catatonia.
Alzheimer [10]	1897	None.	(1) Glial fibrosis..... (2) Degeneration of the ganglion cell body and nucleus. (3) Regressive pigment changes in the body of thickly arranged neuroglial cells.	.....	Positive findings correlated with catatonia.
Alzheimer [14]	1913	None.	(1) Sclerosis of the ganglion cells in the second and third cortical layers. (2) Lipoid degenerative changes.	.....	The general picture of "dementia præcox."
Hassin [28]	1918	None.	Damage as (above) to the ganglion cells of the second and third cortical layers.	These findings are "not uniform, typical or specific for dementia præcox, since similar changes are to be found, sometimes even more pronounced in any other mental disease or organic brain lesion."	None.

TABLE I.—Continued.

TABLE I.—CONTINUED.

Investigator.	Year.	Positive findings.		Negative findings.	Clinical correlations.
		Gross changes.	Microscopic changes.		
Southard [15, 16, 17]	1914-1919	Subcortical areas of gliosis.	"Gliosis" in the supracortical and in the infracortical layers.	.....	Direct clinical correlations: (1) "Delusions are, as a rule, based on frontal lobe disease." (2) "Catatonic symptoms are, as a rule, based on parietal lobe disease." (3) "Auditory hallucinosis is, as a rule, based on temporal lobe disease." [17]
Mott [24]	1922	Case of "advanced dementia præcox"—"very marked congestion and redness of the brain, with minute hemorrhages especially in the medulla."	(a) Changes in the cortex: 1. "Nuclear swelling, lipoid granules, replacing the Nissl granules." 2. Glial proliferation especially in the supragranular layers. Similar changes in the basal ganglia, pons, and medulla. (b) Changes in the reproductive organs: testes: relatively few spermatids. Nuclei of spermatocytes show no mitosis.	.....	Correlation between "primary genetic inadequacy," lack of vital durability, and dementia præcox."
Buscaino [29]	1922	None.	"Multiple areas of disintegration—first in the white substance of the brain, then in the basal ganglia (thalamus), *rarely in the cerebral or cerebellar cortex.*"	.....	Catatonic manifestations are correlated with lesions in the corpus striatum (globus pallidus) and cataleptic phenomena with cerebellar lesions (dentate nucleus).
Dunlap [26]	1924	None.	The "classical findings" are reported in normal controls.	No substantial losses in the outer cortical layers were found after careful cellular counts. Excessive lipoid content found in "normal controls."	None.

TABLE I.—CONTINUED.

Investigator.	Year.	Positive findings.		Negative findings.	Clinical correlations.
		Gross changes.	Microscopic changes.		
Josephy [21]	1926	None.	(1) The third layer is particularly involved (then the fifth); (2) the falling out of ganglion cells is independent of vascular changes; (3) *there is no intrinsic glial response.*	No changes found in the choroid plexus. No significant endocrine findings.	No satisfactory correlation made with the classical entities in dementia præcox.
Bouman [22]	1928	None.	*There is no "selective" process present.* A diffuse spotty involvement due to a primary parenchymatous process involving layers 3, 2, 5, 6, 4 (respectively), characterized by sclerosis and lipoid changes.	"The endocrine findings are of secondary significance."	Changes in the striatum are tied up with hyperkinesis and flexibilitas cerea. Lesions in the temporal lobe (chiefly fields 41, 42 are associated with clearly defined auditory hallucinations. "I can't decide just how far the hebephrenic and catatonic forms coincide with an extension of the process into the frontal and prerolandic areas."
Fünfgeld [48]	1927	None.	Atrophic (Schwund) ganglion cell changes (in layers 3, 5; then 2, 3)—glial reaction (in layer 6).	"Such findings are never found in their entirety in any form of intoxication."	No satisfactory correlation made with the classical entities in dementia præcox. Amœboid glia are found in cases of severe excitements.
Spielmeyer [23]	1930	None.	Ganglion cell loss in the third layer of the cortex, as well as in the deep layers; enormous fat accumulations; regressive changes in the glia.	"We find such indefinite cellular losses in entirely different psychoses."	None.

may be "an elective vulnerability of the parts without any influence of vascular distribution." The work of Brinkman is also cited, as suggesting that cardiac insufficiency may be the underlying

TABLE II.

THE SPECIFIC ORGANIC FINDINGS IN CASES OF DEMENTIA PRAECOX (SCHIZOPHRENIA).

Specific findings.	Hecker.	Kahlbaum.	Alzheimer.	Southard.	Hassin.	Mott.	Buscaino.	Dunlap.	Josephy.	Bouman.	Fünfgeld.	Spielmeyer.
Macroscopic:												
Enlarged ventricles	+	0	0	0	0	0	0	−	0	−	0	0
Hyperaemia of cortex	+	+	0	0	0	+	0	−	0	−	0	0
Hyperaemia of cerebellum	+	0	0	0	0	0	0	−	0	−	0	0
Medullary hemorrhage	0	0	0	0	0	+	0	−	0	−	0	0
Ependymal granulations	0	+	0	0	0	0	0	−	0	−	0	0
Basilar arachnoid exudate	0	+	0	0	0	0	0	−	0	−	0	0
Brain softening	0	+	0	0	0	0	0	−	0	−	0	0
Subcortical gliosis	0	0	0	+	0	0	0	−	0	0	0	0
Microscopic:												
Involvement of Layers II, III	..	..	++	+	−	++	0	−	+	+	++	−
Layers III, V	..	..	+	+	−	+	0	−	++	+	+	−
All cortical layers	..	..	+	+	−	+	0	−	0	+	+	−
Glial fibrosis	..	..	+	+	−	+	0	−	−	+	+	−
Lipoid degeneration	..	..	+	0	−	+	+	−	−	+	−	−
Sclerosis of ganglion cells	..	..	−	0	−	0	0	−	0	+	+	−
Pigmentation of glia	..	..	−	0	−	0	0	−	0	+	+	−
Cortical white substance	..	..	0	0	0	0	++	0	0	0	0	0
Amoeboid glia	..	..	+	0	0	0	0	0	0	0	−	0
Choroid plexus (Von Monakow)	..	..	0	0	0	0	0	0	−	0	−	0
Special Localizations:												
Testes: Decreased mitosis of spermatocytes	..	..	..	..	..	+	..	..	−	−	..	..
Fewer spermatids	..	..	..	..	..	+	..	..	−	−	..	..
Parietal lobe (catatonia)	..	..	0	+	0	0	0	0	0	0	0	0
Temporal lobe (auditory hallucinosis)	..	..	0	+	0	0	0	0	0	+	0	0
Frontal lobe (delusions)	..	..	0	+	0	0	0	0	0	0	0	0
Corpus striatum (catatonia)	..	..	0	0	0	0	+	0	0	+	−	0
Dentate nucleus (catalepsy)	..	..	0	0	0	0	+	0	0	0	−	0
Pons	..	..	0	0	0	+	+	0	+	0	−	0
Thalamus	..	..	0	0	0	+	+	0	+	0	−	0

+ Accepted as a "Specific finding."
− Rejected as a "Specific finding."
0 Not reported.

factor in laminated softening. Bodechtel (1928) (also cited by De Vries) has demonstrated extensive laminated loss of ganglion cells "especially in the third layer in a patient who died 14 hours after temporary heart failure, during narcosis." De Vries sums

up the literature in these words: "The localization in the majority of the cases in the literature and in mine has been in the third cortical layer, but in others it was different or varied in some places. It might be possible that the momentary condition of the vascular system at the time of action of the disturbing agency plays an important rôle in the localization of the lesions." [39]

The suggestions from all of the above authors appear to point in one definite direction—that of the circulation. The theories which include the part played by the blood pressure, cardiac insufficiency, and "elective vulnerability" are brought into a sharp focus by the clinico-pathological investigations of Spielmeyer (1930) and the physiological researches of Forbes and Wolff (1928) on the vasomotor control of the cerebral vessels. The latter investigators have demonstrated by rather ingenious and well-controlled series of observations that there exists a functional activity of the vasomotor fibers in the blood vessels of the pia mater; and have measured the variation in this activity by direct micrometry and photomicrography. Furthermore, they have obtained evidence of a quantitative nature under different conditions.[40]

We, therefore, begin our next series of observations after having established the fundamental activity of the vasomotor fibers of the cerebral cortex; and take another step forward in the solution of the mechanism underlying the lesions which are localized to specific layers when we review Spielmeyer's contribution to this topic. This investigator [41] reports the presence of focal areas of degeneration and linear necrobiotic changes in a series of cases, all of which include cardiac insufficiency. He points out that the same findings (stasis, cellular clumping and slowing down of the flow) occur in the cortical vessels as well as in the peripheral vessels during agonal states, and that these changes are to be considered as the associated phenomena of a vasomotor alteration. Spielmeyer reports cases of arteriosclerosis with or without any demonstration of vessel obliteration in which areas of focal degeneration are to be found.

Spielmeyer concludes from all of these observations that "these findings contribute little or nothing to a differential diagnosis of any special process going on," as the same findings occur in "trauma, carbon monoxide poisoning, morphine and chloroform intoxication, hypertension, pseudouræmia, ecclampsia, emboli, and in cases of post-concussion." These findings present no character-

istic anatomical disease entity but only a "histopathological and pathological symptom complex which, as a rule, follows a central disturbance of circulation due to various noxæ. So that many factors may bring about a similar pathophysiological process and end in a like tissue result."

When Spielmeyer applies these principles to the histopathology of the psychoses, he carries over the lesson which was learned above. He states that there are diseases in which the changes following circulatory alterations are not the expression of the disease process *per se*, but that they are only the expression of certain physiological processes which in turn are the cause of certain symptoms. For example, the localized scleroses in Ammon's horn in the cases of epileptic convulsions are not at the bottom of the disease process underlying epilepsy, whatever it may be; because such sclerosis is also frequently reported in those cases with convulsions in a totally different category; *i. e.*, that of Huntington's chorea. More recently Merrit reports the same findings in the status epilepticus occurring in dementia paralytica.[42]

Neuberger [43] reports the presence of linear softenings of specific cortical layers in a series of cases with arteriosclerotic brain disease. Here also there is little to be seen macroscopically and the author places the falling out of cells on the basis of an ischæmic necrosis occurring probably just before death, and frequently involving the third layer of the cortex. The mechanism of such a process to this author is essentially that of a "vasomotor disturbance, most probably an angiospasm of the smaller vessels, and in general a functional vascular change." In all of his cases there are no thromboses or emboli to be found. As the most reasonable basis for these changes, Neuberger mentions "the architectural peculiarity of the single layers of the cortex in regard to the anatomy and physiology of their small vessels." As yet there are practically no such data. Cobb [45] has demonstrated the absence of end vessels in the cerebral cortex but the detailed working out of the blood supply to the individual cortical layers and their anatomico-functional control is a problem for which we have no data at present.

Neuberger, however, does more than offer an hypothesis for the occurrence of these focal lesions. In another series of observations [44] upon cerebral fat and air emboli he finds a laminated falling of cells occurring in particular cortical layers. But there appears

no special vulnerability of any particular layer; thus the greatest loss of cells may occur at one time in layers 2 and 3, in other cases in layers 3 and 4, and in still others the greatest damage is found in the deeper cortical layers. (Bouman made the same comment in regard to his findings.) [82] Here again, Neuberger feels that we are dealing with a circulatory phenomenon and does not consider it necessary to include Vogt's concept of pathoclysis (to which Bouman would also agree).[82] The tendency of the focal falling out of cells to localize in the second and third layers after embolic phenomena appears to Neuberger to indicate that there is a poorer vascularization in the other layers. At present we have very few facts concerning this topic. The best statement which sums up our knowledge of the relations between the blood supply and the cellular groupings comes from Pfeiffer.[49] He states that "the angioarchitecture does not follow the cellular groupings and course of the fibers in every and each detail, but possesses its own characteristics which have not been investigated completely. Indeed there are angiotectonic pictures which resemble the cyto-architectonic pictures of the same regions as closely as mirror images, but there also exist far-reaching differences between the two structures" (p. 136).

Winkelman in a very recent communication [50] has attempted to supply some of the facts which are necessary before we can come to a better understanding of the relationship of the brain lesions which have been reported in schizophrenia to the clinical pictures of this disorder. This author accepts the brain lesions as "the structural basis of the mental symptoms of schizophrenia" but he clearly states that "there is no characteristic pathologic picture." In this paper he has stressed the importance of the small blood vessels of the brain in psychiatric problems. He has recalled to mind the fact that in cases of endarteritis syphilitica both Nissl and Alzheimer (1904-1905) have described clinical pictures "which resembled, at times to a remarkable degree, those seen in schizophrenia." The changes associated with these clinical pictures have been a "universal mild ganglion cell degeneration with secondary gliosis, with the production of minute acellular areas, ischæmic in nature, the result of focal anoxæmia." The author has observed the same findings in a case of food intoxication. The patient was six years of age and developed a "mild delirium and occasional

convulsions." Winkelman has made the same histopathological observations in the brains of patients who have died during or after acute infections and in a variety of toxic conditions. This author is of the opinion that "it is impossible to recognize from these sections (cases of paresis resembling dementia præcox) the mental and physical make-up of the individual from whom they came." . . . . "*One must, therefore, know the basic make-up of the individual or his personality in order to determine the type of reaction which will occur under the various stresses and strains.*"

Here we have an effective realization of the experimental concept as applied to pathology which has been outlined in a previous section (V). The observation of changes in the brain which are expressed in the clear-cut terms of cellular alteration studied in conjunction with the personality of the individual from whom it came, includes a survey which will prove to be more valuable than any purely "psychogenic" *or* "structural" approach in coming to an explanation as to "what went wrong and why."

When we sum up the data from the observations of Forbes and Wolff,[40] De Vries,[39] Spielmeyer,[41] and Neuberger,[43, 44] we cannot avoid the conclusions that many of the findings reported by many of the previous investigators were the result of just these vasomotor changes which we have described; *i. e.*, Josephy's cases[32] of asphyxia (Case 1), cardiac insufficiency (7), as well as the cases of influenzal pneumonia (9) are to be considered in this category. Moreover, we must begin to see how circumspect we must become in evaluating the effect of all terminal infectious states (as in Mott's case of death following erysipelas).[24] Finally the realization must come that functional vasomotor activity must not only play a great rôle in producing the focal falling out of cells in the supracortical layers, totally independent of any schizophrenic process; but that the investigations of various workers reveal histopathological findings in a great many of the intoxications as well as in cases of circulatory failure which simulate to a marked degree the "specific pathology" described for the schizophrenic disease process.

Recapitulation.

We have gone a long distance both in time and in breadth of concept from the statement made by Kahlbaum in 1874, that we are dealing with a clearly defined "disease of the brain" which

represents a pathological entity.[6] We must be content with much less enlightenment on the schizophrenia-dementia præcox controversy than our immediate predecessors, who, with Gurd,[47] would have agreed "that it seems almost a work of supererogation to-day (1920) to present a study of the organic basis of dementia præcox, since from the infancy of histological neuropathology all skilled observers have been united in recognizing certain essential changes in the central nervous system in cases of dementia præcox." Nor can we agree with this observer, when the statement is made that "all of the great names in neuropathology, each in turn has contributed a histopathological picture of dementia præcox, differing only in the findings but in no case reporting contradictory results" (p. 181).[50]

We have found contradictory results and have attempted to demonstrate that as the observations of the later investigators became more exacting in that they were subjected to controls, and the taking into consideration of the factors of age, terminal conditions, time of autopsy and the type of clinical material with which they were dealing, the claims of the specificity of the earlier findings were progressively decreased. There are, however, careful investigators who still claim that there is a progressive parenchymatous degenerative process of the cerebrum to be observed in cases of schizophrenia (Josephy,[31] Bouman,[32] Spielmeyer,[33]). Nevertheless, in the best of these observations, one which has been carefully controlled by cellular counts in the various cortical laminæ, there is no statement concerning the specific involvement of any particular layer or even series of layers; rather it appears that "the cortex is involved in all of its lawyers, here more, there less" (Bouman [32]).

Again there is a growing consensus of opinion, even among those who insist upon an "organic basis" for this disorder, that these "indefinite cellular losses" may occur in "entirely different organic conditions" (Spielmeyer [33]). On the other hand, the pioneer work of Dunlap [30] has shown how normal controls may demonstrate "many of the so-called classical findings," and that "nobody knows what changes may occur in the nerve cells of normal persons as a result of processes going on in the body just before or just after death—processes in no way connected with any psychosis"—an observation which has been well corroborated by many other

workers (Spielmeyer,[41] De Vries,[39] Neuberger,[43, 44]). *The entire problem is complicated by the existence of a group of progressive degenerations which involve the grey matter of the cerebrum, and from within which the pattern of many of the deterioration reaction may have been taken.* (Alpers.[46])

In conclusion, it appears that we shall have made a definite step forward when we have surrendered the paradigm of general paralysis. After 50 years of intensive research, it has been progressively less productive of constructive formulations as to what is "wrong" with a heterogeneous group of patients which comprise the greatest part of our state hospital population. In the light of all that has gone before, we can only state with any degree of assurance that it is "the picture as a whole (which) makes the diagnosis. There are no pathognomonic findings. The deterioration gives the disorder its name; but it is not always realized" (Meyer[13]). For the present specific anatomical findings and hypothetical toxic influences have proven to be of little value to the clinician, but there is still the patient to be dealt with. What we shall do with the facts which he presents is our major problem and always will be. It is to these facts—of the patient's doings and his sayings—that we should turn our full interest. Whatever features we can best describe (*but always in their individual settings*), let us collect, classify, and try wherever possible to lay down principles which will afford the best possible explanation of most of the facts at hand. Although every type of approach is effective to some degree and no method of obtaining facts can be discounted, this method is the scientific approach, which appears to be most productive of results for the advancement of psychiatry and in the interest of the patient, at the level of our present-day knowledge.

Summary.

An examination of the clinico-pathological evidence offered for the concept of dementia præcox as a specific disease entity presents the following conclusions:

1. There are many contradictory opinions expressed on the topic of a uniform anatomical basis to this disorder. There has been no satisfactory correlation made between the so-called classical groups of dementia præcox and specific anatomical findings.

2. The same *histopathological* findings which are reported as being specific for these groups are also reported as being present in a variety of organic and toxic conditions.

3. There is no uniformity to be found in the various *clinical* contributions as to what constitutes a dementia præcox reaction.

4. The attempt to group these heterogeneous findings into a specific disease entity comparable to that of general paralysis has proven to be progressively less productive of constructive formulations over a period of 50 years of intensive research.

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A NOTE ON A "PROTEOSE-LIKE" SUBSTANCE IN SPINAL FLUID.

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INTRODUCTION.

The investigation of the spinal fluid has been confined almost entirely to a routine examination of known reactions. It is the purpose of this paper to present in a preliminary report the description of a new reaction and substance in the spinal fluid, the presence of which, it is felt, is of sufficient importance to warrant its announcement.

METHOD.

The spinal fluid was obtained from patients admitted to the Colorado Psychopathic Hospital. This fluid was collected in sterile test tubes and kept in an ice box until used.

The fluid was measured and then made acid to congo red by the addition of 25 per cent sulphuric acid. It was then transferred to a separating funnel and thoroughly shaken under moderate pressure with ether, at room temperature, until the ether no longer separated as such. (In the case of spinal fluids of 60 c. c. or more, the quantity of ether used in each instance was 60 c. c. In spinal fluids measuring between 1 and 5 c. c., the amount of ether used was equivalent to two-thirds of the volume of spinal fluid.) The mixture was then allowed to stand. A waxy-gelatinous layer separated at the top. The underlying fluid was drawn off and discarded. To the waxy-gelatinous layer was added absolute alcohol with constant agitation of the mixture. (In the case of spinal fluids of 60 c. c. or more, the quantity of alcohol used in each instance was 50 c. c. In spinal fluids measuring between 1 and 5 c. c., the amount of alcohol

used was equivalent to three times the original volume of the spinal fluid.) A flocculent material separated from the mixture and when allowed to stand, settled to the bottom of the funnel and could be easily removed. This method of extracting the flocculent material which represents the crude proteose-like substance, is referred to in the paper as the ether-alcohol reaction.

Discussion.

The spinal fluids from 37 unselected cases were studied. These fluids were both normal and pathological. In all of them the ether-alcohol reaction was positive and a proteose-like substance isolated regardless of the quantity of spinal fluid used. No relationship was able to be established between this reaction and the amount of spinal fluid protein or sugar or between this reaction and the positive or negative Wassermann or gold sol reactions. The ether-alcohol reaction was present irrespective of whether these chemical substances or reactions were normal or pathological.

Summary.

An ether-alcohol reaction in the spinal fluid is described. The end-product of this reaction is a flocculent material possessing the properties of a proteose-like substance. This proteose-like substance is present in all spinal fluids examined.

A STUDY OF THE TOTAL PROTEIN OF THE CEREBROSPINAL FLUID IN UNCOMPLICATED AND UNTREATED NEUROSYPHILIS.

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Introduction.

The cerebrospinal fluid under normal conditions has been described by Halliburton [1] as being "clear as water and of low specific gravity; it contains in solution inorganic salts similar to those in blood plasma, a trace of coagulable protein, and a certain amount of a reducing substance. . . . " It is this "trace of coagulable protein" with which this paper is specifically interested.

The types of protein present in the fluid according to Hammersten [2] and others are globulin and albumose while Halliburton [3] thinks that they are principally nucleoproteins. Mestrezat [4] identifies the protein as "globulins and faint traces of albumin." The methods of determination of these protein fractions were first devised by Pandy,[5, 10] Nonne and Apelt,[6] Ross and Jones,[7] Noguchi,[8, 12] Weichbrodt [9] and Nonne.[11] Because of the difficulty in differentiating normal from abnormal quantities of precipitated protein and the still greater difficulty of recording the results of these tests for subsequent comparison in different fluids, these methods have been gradually discarded. Quantitative procedures capable of total protein precipitation replaced them because the quantity of precipitated total protein could be reduced to numerical figures which were of definite value and more accurately compared.

Review of Literature.

In the normal cerebrospinal fluid obtained by lumbar puncture the range of the total protein found varies somewhat with the worker. Fremont-Smith and Ayer [13] in 13 cases found the range to be 20 to 40 mg. in 100 c. c. with an average of 28 mg.; Ayer and Foster [14] in 48 cases, 16 to 38 mg., average 25 mg.; Ayer and

Solomon,[15] 30 mg.; Mestrezat,[16] 46 cases, 6 to 32 mg.; average 19 mg.; Denis and Ayer,[17] 35 to 100 mg.; Eskuchen,[18] 20 to 30 mg.; Young, Bennett, Christlieb and Myers,[19] 23 cases, 30 to 80 mg., average 52 mg.; Riser,[21] 50 cases, 15 to 25 mg., average 20 mg.; Penfold and Irving,[22] 9 cases, 10 to 30 mg., average 24 mg.; Mestrezat,[23] 15 to 30 mg.; Ling,[24] 6 cases, 18.8 to 37.9 mg., average 25.5 mg.; Hewitt,[25] average 30 mg.; Grey,[26] 69 cases, 20 to 40 mg., average 28 mg.; Greenfield,[27] 20 to 35 mg.; Fremont-Smith, Ayer, Kennard and Dailey,[28] 13 cases, 20 to 40 mg., average 28 mg.; Bauer and Schenck,[29] 17 to 50 mg.; McNaught,[30] 23 cases, 17 to 40 mg., average 29 mg.; Nawratzka,[34] 15.8 to 28.1 mg., average 22.1 mg.; Quincke,[35] 0.2 to 0.5 mg.; Rieken,[36] 0.5 to 1.0; and Kafka,[41] .02 per cent.

In syphilis of the central nervous system there is rather a disparity of data on cerebrospinal fluid protein. Greenfield [27] states that the cerebrospinal fluid total protein ranges from 40 to 100 mg. per 100 c. c. of spinal fluid. Hewitt [25] in 17 cases finds a range of 60 to 226 mg. with an average of 108; Solomon,[31] 50 to 150 mg., average 89 mg.; Matz,[37] 16 cases, 63 to 204 mg., average 110; Ravaut and Laignel-Lavastine,[33] 70 to 90 mg., average 80 mg.; Denis and Ayer,[17] 200 to 600 mg.; Ayer and Foster,[14] 7 cases, 50 to 153 mg., average 89 mg.; Fremont-Smith and Ayer,[28] 25 fluids, 55 to 200 mg., average 119 mg.; and McNaught,[30] 4 cases, 70 to 116 mg., average 90 mg. In tabes, Greenfield [27] found the cerebrospinal fluid total protein to be 30 to 60 mg.; Hewitt,[25] 6 cases, 48 to 139 mg., average 78 mg.; Matz and Novick,[32] 20 cases, 39 to 87 mg., average 62 mg.; Denis and Ayer,[17] 100 to 200 mg.; Ayer and Foster,[14] 31 cases, 22 to 150 mg., average 78 mg.; and McNaught,[30] 10 cases, 48 to 155 mg.; average 77 mg.

Method of Securing Material.

Any patient, upon admission to the Colorado Psychopathic Hospital, who presented clinical signs or symptoms indicative of pathology of the central nervous system received a lumbar spinal puncture. The cerebrospinal fluid was drawn into sterile, dry, stoppered test tubes, 13 mm. x 150 mm. for subsequent laboratory analysis which consisted of colloidal gold curve test, Wassermann reaction, sugar and protein determinations. The fluid for the protein determina-

tion was placed in an ice box as soon as possible and kept there until the analysis was performed. All contaminated fluids were discarded.

The spinal fluid total protein was determined by the nephelometric method of Denis and Ayer,[17] whereby a 5 per cent sulfosalicylic acid is used to obtain a colloidal suspension of the protein. According to the originators of this method, it is accurate to within approximately 5 per cent.

The cerebrospinal fluids used in this study were from those cases which presented definite clinical symptoms and signs of neurosyphilis and in whom the clinical phenomena were further reenforced by a positive cerebrospinal fluid Wassermann reaction and a positive cerebrospinal fluid colloidal gold reaction. Moreover these cases to fulfill the requirements of the work were untreated and uncomplicated by any pathology other than neurosyphilis which might alter the cerebrospinal fluid.

The cerebrospinal fluid total protein values were handled entirely by means of a statistical technic which was judged to be the most scientific in dealing with the obtained data.

Results.

The cerebrospinal fluid total protein estimations are presented in Table I. This table shows the detailed distribution of the

TABLE I.

No. mg. total protein per 100 c. c. cerebrospinal fluid.	Total no. cases of neuro-syphilis.	Cases of general paresis.	Cases of tabes without psychosis.	Cases of cerebrospinal syphilis with psychosis.
10	2	1	..	1
11	1	1	..	..
12	1	..	1	..
15	4	1	2	1
17	1	..	..	1
20	10	6	1	3
23	3	2	..	1
25	22	12	6	4
26	2	2	..	..
27	1	1	..	..
28	4	1	1	2
30	10	9	1	..
33	3	2	1	..

TABLE I.—CONTINUED.

No. mg. total protein per 100 c. c. cerebrospinal fluid.	Total no. cases of neuro-syphilis.	Cases of general paresis.	Cases of tabes without psychosis.	Cases of cerebrospinal syphilis with psychosis.
35	9	6	2	1
36	2	2	..	..
37	1	1	..	..
38	7	6	..	1
40	21	19	1	1
41	1	1	..	..
42	1	1	..	..
43	3	2	1	..
44	1	1	..	..
45	18	15	3	..
45	1	..	..	..
48	2	2	..	..
50	27	20	5	2
51	1	1	..	..
52	3	2	..	1
53	7	6	1	..
55	15	11	2	2
56	2	2	..	..
57	2	1	1	..
58	6	5	..	1
60	18	14	2	2
62	1	1	..	..
63	7	5	2	..
64	1	..	..	1
65	16	13	1	2
66	2	2	..	..
67	1	1	..	..
68	8	8	..	..
70	21	16	1	4
72	2	1	1	..
73	2	2	..	..
74	1	..	1	..
75	20	16	3	1
76	2	2	..	..
78	1	1	..	..
80	13	12	..	1
83	1	1	..	..
85	4	3	..	1
86	1	..	..	..
88	2	2	..	..
90	20	15	1	4

TABLE I.—CONTINUED.

No. mg. total protein per 100 c. c. cerebrospinal fluid.	Total no. cases of neuro-syphilis.	Cases of general paresis.	Cases of tabes without psychosis.	Cases of cerebrospinal syphilis with psychosis.
92	1	1	..	..
93	1	..	..	1
95	3	3	..	..
96	1	..	1	..
98	2	2	..	..
100	16	15	..	1
105	4	4	..	..
108	2	1	..	1
110	5	5	..	..
113	2	2	..	..
115	1	1	..	..
116	2	2	..	..
120	10	9	..	1
125	1	1	..	..
130	9	5	4	..
132	1	1	..	..
135	4	3	1	..
140	4	4	..	..
150	15	14	1	..
157	1	1	..	..
165	1	1	..	..
170	2	2	..	..
173	2	1	..	1
180	4	4	..	..
192	1	1	..	..
195	2	2	..	..
200	1	1	..	..
201	1	1	..	..
210	4	4	..	..
240	1	1	..	..
300	3	3	..	..
350	1	..	..	1
390	1	1	..	..
420	1	1	..	..

cerebrospinal fluid total protein estimated in the number of milligrams in 100 c. c. of cerebrospinal fluid with the corresponding number of cases in any milligram interval in each type of neurosyphilis.

Table II shows the comparative statistical results obtained.

TABLE II.

Statistical constants.	Neuro-syphilis.	General paresis.	Tabes without psychosis.	Cerebro-spinal syphilis with psychosis.
Number of cases	446	357	48	44
Range	10-420	10-420	12-135	10-350
Mean	75.4	79.0	57.9	64.68
Probable error mean	± 1.62	± 1.86	± 3.0	± 5.08
Standard deviation distribution	±50.99	±52.34	±30.9	±54.0
Q (middle 50% cases)	±34.39	±35.30	±20.84	±33.72

Table III shows the degree of statistical significance of the difference, *i. e.*, chances that true differences exist between the means

TABLE III.

Significant difference between.	Means.	D.	D/P. E. diff.	Chances in hundred.	Statistical significance.
Neurosyphilis and general paresis.	75.40 / 79.00	3.6	1.44	84	None.
Neurosyphilis and tabes without psychosis.	75.40 / 57.90	17.5	5.13	100	Exists.
Neurosyphilis and cerebrospinal syphilis with psychosis.	75.40 / 65.68	9.7	1.82	89	None.
General paresis and tabes without psychosis.	79.00 / 57.90	21.1	5.97	100	Exists.
General paresis and cerebrospinal syphilis with psychosis.	79.00 / 65.68	13.32	2.46	95	None.
Tabes without psychosis and cerebrospinal syphilis with psychosis.	57.90 / 65.68	7.78	1.31	81	None.

of the cerebrospinal fluid protein in the various types of neurosyphilis.

Table IV shows the percentage of cases in each group which

TABLE IV.

Group.	Total no. of cases.	No. cases below 39.99.	Percent of cases below 39.99.
Neurosyphilis	446	82	23.09
General paresis	357	74	20.72
Tabes without psychosis	48	14	29.16
Cerebrospinal syphilis with psychosis	44	15	34.09

fell within the limits of normal cerebrospinal fluid protein. These limits were from 0.00 through 39.99. Any fluid possessing a cerebrospinal fluid total protein above 39.99 was considered pathological.

Discussion.

One of the most important examinations in the fractionation of the cerebrospinal fluid is that of the protein. While normally present in small amounts, an excess of this protein is probably always pathologic in its significance. To be sure, diagnosis seldom rests solely on the protein determination, but when this substance is considered in connection with other tests upon the cerebrospinal fluid, a knowledge of the total protein content is of added value in differential diagnosis. It is of such importance in this respect that the total protein estimation of the cerebrospinal fluid should be used as a routine measure in cerebrospinal fluid examinations and considered of equal importance with the other studies performed.

The origin of the protein normally present in the cerebrospinal fluid is largely a matter of speculation, and until quantitative differential albumin and globulin tests are available, its exact nature cannot be accurately determined. It is well known that there is a progressive increase in the total protein content from the ventricular region through the cisternal to the lumbar region. The chlorides and non-protein nitrogen normally have the same values in these three loci, while the content of the reducing substances diminishes slightly from the ventricular to the lumbar fluid. The reason for these variations has not been determined but it is probable that they are dependent, in part at least, upon the fact that the ventricular fluid is derived almost wholly from the choroid plexus, only a minimal portion coming from the perivascular spaces, while by the time the fluid reaches the lumbar region an appreciable number of perivascular spaces emptying fluid into the subarachnoid area from the brain and cord have been added. The arachnoid cells, lymph spaces within the nerve roots, and the central canal of the cord may, in addition, be sources of this increasing protein. It is possible that the protein present in normal ventricular fluid comes only through the choroid plexus from the plasma, or that it is a product of the ependymal cells. It is also possible that it represents the minimal portion of perivascular fluid which enters the ventricles. But in any event, the total amount of protein is such that the fluid may be considered practically as protein-free and the choroid plexus normally as a membrane impermeable to it.

Due to the lack of adequate and reliable information as to the origin of the protein normally found in the cerebrospinal fluid, it is impossible to come to a definite conclusion in regard to the source of the increased protein found in pathological states such as neurosyphilis. All of the possible sources mentioned for normal protein and in addition transudation through dilated meningeal vessels and tissue destruction may be responsible; this is purely speculative.

It is adequately recognized, though, that in neurosyphilis there is found an increase of the total protein of the cerebrospinal fluid. Halliburton [37] states "under various abnormal conditions the protein matter may be largely increased . . . ."; Ayer and Foster [14] find that "an increase in protein is probably the earliest and most constant abnormality noted in the cerebrospinal fluid under pathological conditions"; Widal, Sicard and Ravaut [38] suggest that an increase in the albumin content of the cerebrospinal fluid is of significance in syphilis of the central nervous system. Grinker [39] states that in neurosyphilis an increase in globulin is found in the cerebrospinal fluid. Fremont-Smith, Ayer, Kennard and Dailey [28] state that "any fluid with a protein content of 60 mg. per 100 c. c. is probably abnormal, and values between 45 and 60 mg. in a large majority of cases accompany central nervous system pathology." Others present opinions of the same order.

In this work 39.99 mg. has been accepted as the upper limit of normal cerebrospinal fluid total protein. It is interesting that approximately one-fifth of the cases of neurosyphilis studied had a cerebrospinal fluid total protein within this normal range.

The mean value for the cases studied fell far beyond the limits of normal as did the middle 50 and 68.26 per cent of the cases.

Another point of interest is the fact that there exists a very definite and unquestioned statistical difference between the obtained means of general paresis and tabes without psychosis, and neurosyphilis and tabes without psychosis. The exact cause of this difference was not ascertained but it is of sufficient importance to be investigated. It might very well be noted, however, that the mean value for tabes fell far nearer the accepted normal range than did the mean value of any of the other types of neurosyphilis.

Summary.

A study of the cerebrospinal fluid total protein measured in mg. per 100 c. c. of cerebrospinal fluid of 446 cases of uncomplicated and untreated neurosyphilis is presented. There were 357 cases of general paresis, 48 cases of tabes without psychosis and 44 cases of cerebrospinal syphilis with psychosis.

The statistical technic was used throughout in evaluating the results.

The average value (M) for cerebrospinal fluid total protein measured in mg. per 100 c. c. of cerebrospinal fluid was 75.4 for all cases of neurosyphilis, 79.0 in general paresis, 57.9 in tabes without psychosis, and 65.68 in cerebrospinal syphilis with psychosis.

The middle 50 per cent ("Q") of the cases had a cerebrospinal fluid total protein falling between 41.01 and 109.79 mg. for all cases of neurosyphilis, between 43.70 and 114.30 mg. in general paresis, 37.06 and 78.74 in tabes without psychosis, and 31.96 and 99.40 mg. in cerebrospinal syphilis with psychosis.

There is no true difference between any of the means excepting neurosyphilis and tabes without psychosis, and general paresis and tabes without psychosis. In these two instances the difference is so pronounced that there is no question concerning its existence.

If 39.99 mg. per 100 c. c. of cerebrospinal fluid is accepted as the upper limit of normal protein, it was found that 23.09 per cent of all the cases of neurosyphilis possessed normal values. 20.72 per cent of the cases of general paresis, 29.16 per cent of the cases of tabes without psychosis, and 34.09 per cent of the cases of cerebrospinal syphilis with psychosis also possessed normal values.

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THE PSYCHIATRIC RESOURCES OF NEW YORK.

A Brief Description for Those Attending the 1934 Meeting.

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Everybody hopes to visit New York. Every American expects to see New York. Every psychiatrist wishes to come to New York this year and attend the 90th annual meeting of The American Psychiatric Association. Eight years have passed since the Association last honored Gotham by holding its convention here and many of our members must be unfamiliar with the present detail and scope of psychiatry in New York. To such, these notes are particularly addressed. They cover the majority of prominent psychiatric facilities.

In relation to such a gathering we think of New York as a metropolitan region with many psychiatric interests that center on the Island of Manhattan. We hope that visitors from other sections will plan their trips so as to study the psychiatric facilities of this district in a comfortable and leisurely manner. Every institution, public and private, will welcome a visit from anybody that attends these meetings.

We do not forget that our neighboring commonwealths, Connecticut and New Jersey, have hospitals that are worth all the time one can spend at them. Only those that are more easily reached from New York will be mentioned, since others have been discussed in the publications circulated at meetings in other centers. Similarly institutions of central and western New York are more naturally described in relation to meetings in the Buffalo district, to which the Association occasionally goes.

New York would need and doubtless have a fine range and variety of psychiatric facilities merely because of its size if for no other reason. Life in New York is none too gentle in its emotional demands, but nevertheless attractive, and many of those who come here must at some time be given psychiatric help. The

record discloses that even before the War of Independence a humane and intelligent public opinion sought to meet such needs. When changing times and developing therapy have indicated that there is need of some new attack on human problems there has not lacked a group who would find the means, human and monetary, with which to make the new effort. Though many such organizations have been hard tested in the last three years they will be found carrying on their work despite every handicap.

Charles Dickens visited New York as well as Boston in 1842. He could not approve all he saw. This Association had not yet come into existence and there was no authoritative voice on institution standards. He says in "American Notes":

> One day, during my stay in New York, I paid a visit to the different public institutions on Long Island, or Rhode Island: I forget which. One of them is a Lunatic Asylum. The building is handsome; and is remarkable for a spacious and elegant staircase. The whole structure is not yet finished, but it is already one of considerable size and extent, and is capable of accommodating a very large number of patients.
>
> I cannot say that I derived much comfort from the inspection of this charity. The different wards might have been cleaner and better ordered; I saw nothing of that salutary system which had impressed me so favourably elsewhere; and everything had a lounging, listless air, which was very painful. . . . . I have no doubt that the gentleman who presided over this establishment at the time I write of, was competent to manage it, and had done all in his power to promote its usefulness: but will it be believed that the miserable strife of Party feeling is carried even into this sad refuge of afflicted and degraded humanity? Will it be believed that the eyes which are to watch over and control the wanderings of minds on which the most dreadful visitation to which our nature is exposed has fallen, must wear the glasses of some wretched side in Politics? Will it be believed that the governor of such a house as this, is appointed, and deposed, and changed perpetually, as Parties fluctuate and vary, and as their despicable weathercocks are blown this way or that? A hundred times in every week, some new most paltry exhibition of that narrow-minded and injurious Party Spirit, which is the Simoom of America, sickening and blighting everything of wholesome life within its reach, was forced upon my notice; but I never turned my back upon it with feelings of such deep disgust and measureless contempt, as when I crossed the threshold of this madhouse.

The institution visited by Dickens was on Blackwell's Island and was long since discarded. Thankful we are that we have come a long way since 1842!

In the first 50 years of its existence this Association met four times in New York. It was customary then as now for the mem-

bers of the Association to visit institutions in the neighborhood of their meeting place. These visits were taken seriously and the Association did not forebear to go on record if conditions found were unsatisfactory. Such was the case at the meeting in 1848, the first one held in this city. The Association accepted an invitation to visit the asylum on Blackwell's Island; then adopted some resolutions. It would be far more grateful to their feelings, they said, could they leave this institution in silent but respectful regard. They considered it necessary to declare their conviction that the arrangement for the three or four hundred pauper lunatics of this city was far in arrear of the age; they appealed to the authorities of this mighty and opulent metropolis of the western world to sustain the honor of their position. Specifically they implored that the patients should not be abandoned to the tender mercies of thieves and prostitutes who were, to a considerable extent, the associates and keepers of the patients. They objected to "those miserable apologies for insane hospitals, known as the old and the new madhouses." One of the members wrote home that the patients were crowded, unclassified, and in every imaginable condition of degradation, disease and despair. It is probable that the strong, though very courteously worded resolutions of the Association were a source of real help to the medical board that had already protested such conditions, for when the Association again came to New York in 1851 it resolved that it "cannot refrain from expressing the high gratification" of finding a very marked improvement.

Probably the members of this organization, then known as The Association of Medical Superintendents of American Institutions for the Insane, did not see every detailed advance that should be made. The remarks on the Bloomingdale institution were laudatory and doubtless should have been; nevertheless Dorothea Lynde Dix was of the opinion in 1851 that the hospital should not have three heads; that the physicians should not change so often; that there should be a night watch in each lodge; and that other improvements should be made.

At the twelfth annual convention in 1857 there was complaint that the longhand reporter employed the year before had performed his duties in an imperfect manner. There was also allusion to previous difficulties with reporters. Evidently some matters had

reached the public press in unsatisfactory form. It was accordingly arranged that the publishers of the Journal should employ, at their own expense, a competent phonographer to report in full the proceedings and submit them before publication to each member for revision and approval and also to give a brief abstract of the proceedings to the newspaper men.

* * * * * * * * * *

In the descriptive notes that follow, the oldest hospital organization is first discussed. Thereafter a roughly topographic plan is followed, relating each institution to its neighbors. In all directions for travel, the mileage is approximate. To points within the city, it is calculated from the Waldorf Hotel. To points outside the city it is calculated from the railroad terminus. Highway distances are usually figured from Columbus Circle which is less than a mile from the hotel.

The visitor coming from the east is probably well acquainted with the psychiatric resources of New England. Those in Connecticut that he would be most likely to wish to see are: The Connecticut State Hospital at Middletown, The Fairfield State Hospital at Newtown, The Hartford Retreat, The Institute of Human Relations at New Haven, The Mansfield State Training School at Mansfield Depot.

If the visitor prefers to cross the Long Island sound by the ferry at New London or one of those farther west he can visit the hospitals at Kings Park, Central Islip, Northport, Brentwood and Creedmore on his way to the city where the meetings will be held.

Coming from the north one is likely to follow Route 9 or Route 22 as he pushes south from Whitehall. From Route 9 he can easily visit the Hudson River State Hospital and the Matteawan State Hospital. Route 22 takes him by the Wassaic State School and the Harlem Valley State Hospital.

The traveller from the west finds many roads whose terminus is New York. In the Mohawk Valley are the Rome State Training School and the Utica State Hospital. Near the southern edge of the state are the Binghamton State Hospital and the Institution for Defective Delinquents at Napanoch.

If the visitor comes across Pennsylvania he may wish to visit the psychiatric resources of the Philadelphia region or to see the Allentown and Wernersville State Hospitals or the Veterans Hospitals at Coatesville.

Whoever comes through New Jersey can stop at the State Hospitals at Trenton and Greystone Park, The Essex County Hospital at Cedar Grove, or the Veterans Hospital at Lyons.

The Society of the Hospital in the City of New York in America.

Before 1770 there were only two hospitals in the country. One of these was a public institution for the insane in Virginia.

The other was the Pennsylvania Hospital which made provisions for a broad medical service, including the mentally sick. In 1769 the first steps were taken toward establishing an adequate general hospital in New York and on June 11, 1771 a royal charter was granted to the Society of the New York Hospital. From the first it was intended that suitable care should be given to those with mental illnesses. A spacious tract northwest of the present City Hall was the site of the new institution, and construction began promptly. Before any patients were admitted an accidental fire in 1775 gutted the building and necessitated reconstruction. In 1776 the building was again ready but now the Revolution was in progress and the new building was used for barracks, first by the Continental troops and then by the British. After the war the economic disorganization of the city was such that for a few years the hospital lacked funds to organize; in fact just two decades passed from the granting of the charter to the opening of the institution.

In January 1791 the first patient was received in the handsome and commodious building that continued to serve the city for many years. The first mental case was received in September 1792. The records of the hospital indicate that several wards were devoted to the care of "maniacs"; there were quarters in the basement for the isolation of any illness that could not be properly accommodated upstairs but most of the mental patients seem to have been above the ground floor. In 1797 the Board of Governors in defining the policy of the hospital stated that "Persons laboring under incurable decrepitude, or long-continued ailments of any kind, are considered fitter objects for an almshouse than for this hospital, which is properly an infirmary, for the reception of such persons as require:

1. Medical treatment.
2. Chirurgical management.
3. Maniacs, and
4. It is contemplated to fit up a lying-in ward, etc."

The psychiatric service was so useful that the state was ready in 1808 to make an annual appropriation for 50 years in order to place its patients here. At times other public moneys were

granted for specific purposes up to the time of the Civil War. By 1865 the state and county institutions had been developed to a point where public patients could be adequately accommodated and all grants of public money to this institution ceased.

Bloomingdale Hospital.

New York Central Railroad from Grand Central Station, 23 miles. Highway: Central Avenue or Bronx Parkway, 25 miles.

To accommodate its expanding psychiatric service, the Society erected in 1808 a separate two-story building accommodating 75 patients. At this time an attending physician was appointed and for some years the service was directed in the same way as the medical and surgical services. Only gradually was the authority of the resident physician increased and not until 1877 was the chief physician made also the chief executive officer of the institution. Ten years passed after the erection of this building, and it was already inadequate. Under the leadership of Thomas Eddy, an officer of the Society and a member of the Society of Friends, a tract of land was purchased in the country at the seven-mile stone on the Bloomingdale Road. In 1821 the mental patients were moved to the new institution. The magnificent site overlooked both the Hudson and Harlem Rivers. The land was laid out in walks, gardens, groves and playgrounds. The governors published an address to the public in 1821 in which they discussed the new conception of "moral treatment" of the mentally afflicted and dedicated themselves to conduct the institution in that spirit. This building considerably enlarged in later periods, served until 1894. From the name of the thoroughfare the institution was called the Bloomingdale Hospital. Seeing the rapid growth of the city, the Board of Governors in 1868 purchased land in White Plains and to that site the institution was moved in 1894.

The original plant at White Plains has been supplemented by the addition of further housing for patients, but the greatest expansion has been in treatment facilities. The hospital now accommodates somewhat fewer patients than formerly, and in the provision for medical staff, nursing personnel, physical activities and occupation therapy the expansion has been strik-

ing. It has been possible for the institution to do experimental work and to establish standards in certain matters where public institutions could less well take the first steps.

Dr. Pliny Earle, one of the founders of this Association, was resident physician from 1844 to 1849. His successor was Charles H. Nichols. The public patients being now withdrawn, he advised that better provision be made for those of greater means, and out of this recommendation grew the present policy of the hospital to serve primarily the business and professional classes. The Board of Governors from the foundation of the hospital had been especially interested in providing for recoverable patients, and with its increase of treatment facilities, more emphasis has been given to this policy. Bloomingdale Hospital therefore at the present time attempts to make very good provision for a considerable group of patients whose social treatment requires a more liberal expenditure than is feasible at all institutions, and in very many instances is much greater than the relatives can meet without assistance from the hospital funds. In cases of favorable prognosis such assistance is forthcoming. Indeed a very great contribution is made every year toward the cost of care of those whose funds are inadequate.

Payne Whitney Psychiatric Clinic.

Lexington Avenue subway or surface car to 68th Street. Courtesy bus to New York Hospital. 1½ miles.

For more than 100 years the mental patients cared for by the Society of the New York Hospital were at a point several miles from the other services. When the Board of Governors planned the removal of the New York Hospital from West 16th Street to the new site on the East River at 68th Street, they found means through the generosity of the late Payne Whitney to build and equip a psychiatric unit. Mr. Whitney, as vice-president of the Board, had for some years shown a keen and most helpful interest in the activities of Bloomingdale Hospital. Hence the provision in his will for the new establishment was not unexpected. The Payne Whitney Psychiatric Clinic has restored to the department of psychiatry the close

relation to other hospital services that existed in the first 30 years of the hospital's activities.

The clinic is at the right of the main entrance to the hospital group. The location is such that its patients have both accessibility and privacy and the quiet that is so important in treatment. Below the ground level there are two stories fully connected with the other buildings by corridors. Since the building stands on a bluff overlooking the East River, these lower stories have their share of light and fresh air.

The building is U-shaped and the partly enclosed court looks toward the river. The total length is 130 feet and the wings extend 51 and 60 feet respectively. The building has been designed not only for treatment but also for teaching and research. To it come the students of the Cornell Medical School for their courses in psychiatry.

There are 88 beds for adults and 23 for children. The plans of this clinic were very carefully studied so that the provisions available throughout the general hospital would be supplemented by those detailed arrangements that are so desirable in meeting the needs of the mentally sick. Continuous flow baths are available on all the five floors where patients reside. Artificial ventilation and heat control and noise control are provided in the rooms for the restless patients. Windows are specially designed. There is a gymnasium and the roofs are equipped for out-door exercise, and a garden and two enclosed courts are available. Occupation therapy has suitable provision.

State Department of Mental Hygiene.

Lexington Avenue Subway to Worth Street. 3 miles.

In 1873 the Board of State Commissioners of Public Charities were by statute given duties to be performed by a Lunacy Commissioner. He was to examine and report conditions and management and results of treatment. This arrangement for inspection continued until 1889 when a Commission in Lunacy was created consisting of three members, the president of whom would be a physician experienced in the management of institutions for the insane. The title was change to State Hospital Commission in 1921. In 1927 under a reorganization of the

state government the Department of Mental Hygiene was created, headed by a medical commissioner of at least 10 years' practice and five years' institutional experience. The department inherited all the powers and duties of the former commission and received control also of institutions for defectives and epileptics.

The main office of the Department of Mental Hygiene is in Albany but it maintains offices also at 80 Centre Street in the State Office Building in New York City. Here can be obtained information about the varied activities and interests of the greatest organization of this nature in the country. All the mentally sick of New York are cared for by the state, save those who are maintained by their relatives in private institutions and those whose cases are awaiting disposition in some general hospital or minor psychiatric institution. Included in this department are 20 hospitals for the mentally sick, five state schools for the care and training of mental defectives and one institution for the epileptics. The population of the hospitals is now over 50,000, of the schools and epileptic institution 11,000. In addition the department is responsible for inspection and advice for the two institutions for insane criminals and two schools for defective delinquents. It has jurisdiction also over all licensed private institutions, 31 in number, with a population (including two Veterans Administration Facilities) of 2000 patients, and over the 10 institutions licensed for the care and treatment of mental defectives.

The department maintains an inspection service not only over medical matters but also covering all phases of administration. The psychiatric inspection covers not only the institutions belonging to the department but every place where persons suffering from mental disease or defect receive care and treatment. Information on all details of institution management is assembled by the department, since the control of funds appropriated by the state is lodged in it. The commissioner nominates superintendents when vacancies occur and in general sees to it that the provisions of the mental hygiene law are carried out and that the highest possible standards of care and treatment are maintained. The department is responsible for the

development of plans for increasing institution capacity, for devising and adapting the best types of treatment for all its patients, and in general must exercise a very broad, reasonable, but energetic influence on all psychiatric activities of the state.

National Committee for Mental Hygiene.

The National Committee will be in new offices in the Rockefeller Center only a short distance from the hotel where our 1934 meetings are being held. This organization, founded in 1911 through the energy and foresight of Clifford W. Beers, from its inception has enlisted the services of a remarkable number of outstanding laymen and physicians, especially psychiatrists.

Its first efforts were devoted to improving the care of the mentally sick. Soon the committee was called on for advice and assistance in obtaining proper care and education for mental defectives, both through the organization and improvement of state schools and also in the development of special classes and other community facilities. Child Guidance clinics were fostered by this organization. The problems of the prisoner called for study and advice, as did the field of dependency. The various branches of education have sought and obtained much aid, and still other problems have been brought to its attention. Thomas W. Salmon, the first medical director, was a president of this Association.

The National Committee has conducted surveys of the care of the mentally sick, the training of defectives, and other problems of mental hygiene in 34 states. It has played a leading rôle in knitting together the psychiatric forces of the country. In the office will be found plans of some of the best buildings of the state hospitals and schools. The library is available to members of the American Psychiatric Association as well as to other students of the problems of mental health.

For more than a year The American Psychiatric Association has had its headquarters in the offices of the National Committee. The staffs of both organizations are at the service of those who are attending this meeting, either at the Rockefeller Centre or in the hotel.

New York State Committee on Mental Hygiene.

Lexington Avenue subway or surface car to 23d Street. 1½ miles.

This group functions with and as a part of the State Charities Aid Association at 105 East 22d Street. Its purposes are to conserve mental health and to assist in securing high standards of care, treatment and training for those who suffer from mental disease and defect. Its methods are fact-finding, and it disseminates information, promotes plans for the advancement of mental health, and participates in many educational projects. Many local committees are affiliated with the State Committee, one of which is located in New York City. Its policies may well be studied by those who are engaged in similar projects elsewhere.

New York State Psychiatric Institute and Hospital.

Subway at Broadway and 50th Street or Seventh Avenue and 49th Street to West 168th Street. Walk two blocks. 6 miles.

The Psychiatric Institute stands on a bluff called Washington Heights and is the nearest to the Hudson of all the buildings in the Medical Center. This organization was established in 1895 under the title of the "Pathological Institute." At first it had quarters on Madison Square but from 1902 till the erection of the present building in 1929 it was located on Wards Island. The first director, Dr. Ira Van Gieson, was a distinguished neuropathologist who gathered around him a corps of workers who correlated various sciences in the investigation of the problems of mental disease. Dr. Boris Sidis was the associate who directed the psychological inquiries. Dr. Adolf Meyer was the next director, serving from 1902 to 1910. In many regards this was one of the most fruitful periods of Dr. Meyer's professional activity for there came under his influence the whole medical service of the New York state hospitals, men who are now found in all parts of the country. When Dr. Meyer went to Johns Hopkins University he was succeeded by Dr. August Hoch who remained seven years. All those who came under his persuasive personality carried away from that period some of his keen interest in psychogenic factors and personality trends. He was succeeded by our president,

George H. Kirby, who was medical director from 1917 to 1931. He had the pleasure of dedicating the present building and of organizing the additional activities that then became possible.

The work carried on by the Institute is indeed varied in character and closely correlated with the medical service of all the state institutions. The types of study that are in progress are themselves memorials to the several psychiatric leaders who have successively directed the institution. Another type of memorial will be found in the lobby of the auditorium in the case of two men who in very different ways had a pronounced influence on the Institute, and on psychiatric thought. One plaque commemorates Dr. Thomas W. Salmon who originated the idea of moving the Institute from Wards Island to the Medical Center and whose experience and thought were given most generously to planning the building and organization. The other is to Dr. Charles B. Dunlap, one of the outstanding neuropathologists of his time. The Institute has laboratories for work in neuropathology, chemistry and bacteriology, and wards for the accommodation of adults and children of both sexes. Staff members of state hospitals and state schools come here for courses of instruction and sometimes to work out individual research problems. The medical students of the College of Physicians and Surgeons get their courses in psychiatry here. Other miscellaneous teaching functions are carried out on a considerable scale. The personnel includes therefore psychiatrists, dentists, chemists, bacteriologists, psychologists, consultants in many medical fields and the necessary complement of nurses, social service workers, occupation therapists and technicians of many sorts

New York Neurological Institute.

Adjoins the Psychiatric Institute.

While primarily for the study of neurological disorders, this Institute has a service for psychiatric problems. The building is in the Medical Center, carefully studied in all functions, and admirably equipped for its work. Close cooperation is maintained with the medical school and the other hospitals of the Center.

Manhattan State Hospital.

Lexington Avenue subway to 116th Street. Eastbound surface car to ferry. Four miles.

The city of New York began to make definite provision for its mentally sick patients in 1825. It then withdrew patients from Bloomingdale Asylum and removed others from the Almshouse and domiciled them in the basement and first story of a new general hospital on Blackwell's Island. This is the same arrangement that had been used 30 years before at the New York Hospital. They were under the treatment of visiting physicians from Bellevue Hospital until 1847. The care for many years was very unsatisfactory; an attendant appointed by the Common Council had charge of each ward with two convicts as assistants. This however was the beginning of what became an elaborate organization that reached the extent of four branch institutions before the city passed its responsibilities to the state in 1896.

One outgrowth historically of the city organization is the Manhattan State Hospital on Wards Island. Two presidents of this Association, Alexander E. Macdonald and C. Floyd Haviland, were superintendents of this institution and other prominent members such as Doctors Dent and Mabon directed its affairs at different times. For many years this hospital has been a place of great psychiatric activity. Its buildings represent various periods of hospital construction and indeed some of them were originally erected for other purposes; one group was the general hospital for immigrants, another building was the inebriate asylum. The population is almost 4000 and for sometime was considerably larger. Its convenient location has brought to it visitors from all the world, and all types of thought on psychiatric matters have been discussed here.

Hudson River State Hospital.

New York Central Railroad to Poughkeepsie. 74 miles. Highway: Riverside Drive and Albany Post Road. 77 miles.

This institution dates back to 1871 during which year it accommodated seven patients. It was the third to be established by the state, being preceded only by Utica and Willard. It

is related that the cost of the original buildings was considered high and met much official criticism, but they were well constructed and have served two generations well. A former president of this Association, Charles W. Pilgrim, was superintendent of this hospital for thirteen years. The land reaches down to the Hudson River and runs back into the hills, covering a thousand acres. The main building has a magnificent view of the river. Another group of buildings is a mile away. Thus the compactness of the original plant has been relieved by the plan of later development.

Matteawan State Hospital.

New York Central Railroad to Beacon. 58 miles. Highway: Broadway and Albany Post Road to Fishkill; turn left. 67 miles.

The Matteawan State Hospital was our first institution for insane criminals located wholly apart from prison and civil hospital. There had been much complaint because such patients were mingled with others in the older state hospitals; this was based partly on sentiment and partly on the actual perils of association with some aggressive patients. The hospital was opened in 1892 on a beautiful site overlooking the Hudson. The original buildings were constructed to secure a very high degree of protection and ease of supervision and so well were the plans drawn that they have been adapted for institutions in other states. With the growth of the hospital there gradually accumulated a considerable number of patients whose conduct was not so difficult to control and the newer buildings have been planned in accordance with these simpler needs. The institution receives all the psychotic women who are committed from penal and correctional institutions. In the case of men those who are serving sentences of less than a year are admissible here on becoming insane. Persons who without conviction are found in court to be insane are committed to this institution. There is also provision for transfer from civil state hospitals of a few patients who need the type of oversight available here. The census is between 1200 and 1300.

Harlem Valley State Hospital.

New York Central Railroad to Wingdale. 70 miles. Highway: Bronx River Parkway to Valhalla, routes 22 to Armonk, 128 to Mt. Kisco, 117 to Katonah, 22 to the hospital. 74 miles.

In 1924 the Harlem Valley State Hospital was opened to relieve the over-crowding in New York City institutions and to provide as well for some of the population in its neighborhood. The site belonged formerly to the prison department but was never utilized by them. It rises sharply from the valley and spreads over the adjoining hillside; hence there are buildings picturesquely placed on several levels. The institution being new has a coherence about its plan that is not always evident in older institutions. It has already 1700 patients and will be further developed.

Wassaic State School.

New York Central Railroad from Grand Central Station to Wassaic. 82 miles. Highway: Bronx River Parkway to Valhalla. Route 22 to institution. 91 miles.

In 1930 the Wassaic State School was opened, an entirely new structure planned to house 2900 defectives. This capacity has not quite been reached. The school is situated on some hilly slopes and buildings are scattered on several levels. The institution has all the advantages of being completely planned before construction began.

Rockland State Hospital.

Erie Railroad to Orangeburg; or bus from 33d Street and 6th Avenue. Highway: Riverside Drive, George Washington Bridge and Route 9W to Sparkill. 23 miles.

A recently built institution all of whose facilities were planned at the same time is the Rockland State Hospital at Orangeburg, a few miles west of the Hudson. This was planned to accommodate about 4000 patients and is already fully occupied. The buildings are arranged in three groups. One is for the reception service and other acute cases, the plan being that the average residence in this group might be about nine months. For each sex there is also another large group of buildings. These structures are of concrete and generally have low spreading lines resembling Spanish architecture. The window and porch arrangements are interesting.

Letchworth Village.

West Shore Railroad from 42d Street to Haverstraw; taxi. Highway: Riverside Drive, George Washington Bridge and Route 9W to West Haverstraw, then two miles west. 40 miles.

Letchworth Village is a state training school for mental defectives in Rockland County, across the Hudson from New York. It is in the hills with 2100 acres of land ranging from 400 to 1100 feet above sea level. There are several groups of buildings well spaced so that the sexes are separated and the older and younger patients are at an appreciable distance from each other. All the industries of the institution and many additional activities are carried on by the inmates. They are given whatever type and amount of education they can master. Structurally the institution is unusual in that relatively simple one-story buildings have been preferred, and have been adopted for most of the institution. There are 2900 children and adults under care. Additional buildings are under construction.

Middletown State Homeopathic Hospital.

Erie Railroad to Middletown. 67 miles. Highway: West 42d Street ferry, routes 9W and 17. 81 miles.

This hospital was started by the subscription of private funds to the extent of $50,000 and was later completed by the state, the first patient having been received in 1874. The hospital had from its early days the benefit of the special interest of those who were interested in homeopathic practice and at times the public maintenance funds were supplemented by private gifts. With the passage of time considerable additions were made to the plant which now houses 3000 patients. One of the buildings bears the name of Doctor Selden H. Talcott, the best known of the earlier superintendents.

Institution for Male Defective Delinquents.

West 42d Street ferry. N. Y., Ontario & Western Rr. to Napanoch. 95 miles. Highway: Riverside Drive, George Washington Bridge, routes 4, 2, 17 and 279. 96 miles.

In the large prison population of New York State the problems of mental disease were long ago appreciated and provided for. Later it was recognized that a considerable number of

mental defectives in prisons and especially in reformatories are a drag upon any program of rehabilitation, since they cannot be expected to grasp as quickly and act as promptly as do those who have better intellects. Accordingly the state established a special institution for male defective delinquents and opened it in 1921 at Napanoch. In some instances inmates are received from the civil institutions for mental defectives. In other instances transfers are made from reformatories. A large number come on direct commitment from the courts. The institution is so constructed that there are necessary safeguards against escape but in addition it is provided with facilities for industrial training suited to the capacity of those who are under care. The usefulness of the institution is attested by the fact that it always operates with a census considerably above its certified capacity. Almost 1000 inmates are accommodated.

Brooklyn State Hospital.

Lexington Avenue subway to Winthrop Street. Clarkson Avenue to Albany Avenue. 9 miles.

The County of Kings, the largest part of which was then the Town of Brooklyn, built a county poor house and occupied it in 1832. Those insane persons who could not be cared for comfortably in it were boarded in the Bloomingdale Hospital. Five years later the county contracted for the erection of a "Lunatic Asylum and Work House," which was opened in 1838. This building was quite up-to-date, being distinguished by having a shower bath. It was later outgrown and so was its successor but the third such institution, opened in 1854, was the nucleus of what is now called the Brooklyn State Hospital. The original building has been considerably modified and other buildings of quite different type have been added. The institution was turned over to the state in 1895. Twelve hundred patients are cared for here. The service is particularly interesting because of the large number of weak and ill patients. Of those committed to state care from this populous section of the city, the able-bodied are sent out to the country and the feeble are kept here.

Creedmoor Division.

Long Island Railroad from Pennsylvania Station to Queens Village. 10 miles. Highway: Queensboro Bridge to Queens Boulevard, Kew Gardens, Grand Central Parkway, Springfield Boulevard. 11 miles.

A branch of this hospital has grown to a greater capacity than the parent institution. This is the Creedmoor Division with 3000 patients, on a plot of 314 acres. A complete hospital was planned for this site and the new institution has those advantages that come from the relevance of facilities to the needs of the population.

Central Islip State Hospital.

Long Island Railroad from Pennsylvania Station to Central Islip. 43 miles. Highway: Park Avenue, East 57th Street, Queensboro Bridge, Northern Boulevard, and Merrick Road to East Islip. Turn left. 45 miles.

The City of New York (before consolid tion with Brooklyn) maintained a series of mental hospitals on several islands in the East River. For their relief the city purchased in 1887 900 acres on Long Island and two years later placed patients in the first buildings erected there. This institution, taken over by the state in 1896, is now the Central Islip State Hospital. Every few years some new building project has been under way and the hospital has expanded until now the population has risen to 6600. The land is level and originally seemed to be of limited fertility but its intelligent cultivation and redistribution for 45 years has made it a very attractive property. Here is one of the first groups of separate dining rooms clustered around a common pantry.

Kings Park State Hospital.

Long Island Railroad from Pennsylvania Station. 45 miles. Highway: Park Avenue, East 57th Street, Queensboro Bridge, Northern Boulevard. 45 miles. Trip may be combined with visit to Veterans' Hospital at Northport, 5 miles.

When the Kings County Lunatic Asylum was utterly overcrowded in 1885 the county purchased 850 acres of land on the north shore of Long Island for a branch institution. This has grown into the Kings Park State Hospital. Its history illuminates the difference between ordinary county and state

ideals in the care of the mentally sick, and a considerable change occurred when the state took over the hospital in 1896. The old scandals left scars on organization and construction for years. Its course of development has produced buildings of varied type and located in several distinct groups on a pleasant wooded site that reaches from a village by the railroad on one side to Long Island Sound on the other. Perhaps the most interesting of these groups is the one erected for the care of veterans who for any reason could not be accommodated in the government hospitals. The population of the institution is over 4300.

Pilgrim State Hospital.

Long Island Railroad from Pennsylvania Station. 39 miles. Highway: Queensboro Bridge, Northern Boulevard to Huntington and Route 25 to Commack. 41 miles.

This hospital, located at Brentwood is the newest in the New York State system. It will have the distinction of being the largest institution in this region but is by no means yet complete. Already over 4600 patients are quartered here. The concept that a mental hospital may well care for several thousand patients has been accepted in New York somewhat more readily than in smaller communities. The population of the city is quite accustomed to large buildings and small quarters so that even the most crowded of the state hospitals may seem spacious and luxurious to the apartment house dweller of Manhattan. When it became evident that patients must be moved from Wards Island (which once had a population of much over 6000) it was decided to prepare adequate accommodations not only for the patients already under care but for the increasing state hospital census of the next few years by building an institution for 10,000. This hospital was named for a former president of this Association. It is easy of access from New York and on land that presents no important problems for either the architect or the sanitary engineer. The architectural style is Lombardy and all buildings are of red brick, of concrete flat arch construction, with mission tile roofs.

Veterans Hospital.

Long Island Railroad from Pennsylvania Station to Northport. Taxi. 40 miles. Highway: Queensboro Bridge, Northern Boulevard and Route 25A. 40 miles. Trip can include Kings Park State Hospital, 5 miles.

In a picturesque setting on rolling land that includes the highest point on Long Island is the Veterans' Administration Facility at Northport. There are over 27 hospital buildings. Most of them surround a huge oval exercise ground and are connected by corridors or underground passageways. The institution was carefully planned by the construction division of the Veterans' Bureau on the basis of experience with its earlier hospitals and advice from state officials and others well acquainted with the problems. All buildings are fire resistant. Various types of patients have been given the special accommodations demanded by their needs. The emphasis laid by the Veterans' Administration on proper occupation and recreation facilities is well known and can be profitably inspected in this hospital. Especially have the needs of the physically ill, the tuberculous and the disturbed been given particular attention. Administratively the hospital operates under a license from the State Department of Mental Hygiene and is classed among the private institutions. It now cares for about 1400 patients.

Bellevue Hospital.

Lexington Avenue surface car to 30th Street. Walk east to First Avenue. 1 mile.

The city of New York had no provision in any general hospital for the care and treatment of the mentally sick from 1821 to 1879. In the former year the Society of the New York Hospital had moved its mental patients to a separate institution in the country. No other hospital cared for them. Such patients frequently were taken to Bellevue Hospital and when an addition was put on that structure in 1825, 24 rooms including so-called "alcoholic cells" were constructed. There was a definite effort to pass such patients quickly to the city asylum but delay was inevitable and they were thrown into the company of acute alcoholics and criminals. On November 11, 1826, out of a census of 184 the insane numbered 82. Again

during an investigation in 1837 more than half of the 265 patients were found to be insane. It was not until 1839 that they were removed to Blackwell's Island.

In 1879 a special pavilion was erected at Bellevue Hospital for the reception of the insane. There were no facilities for treatment, and intolerable conditions arose from lack of constant supervision and unfit type of nursing force. In 1882 a resident physician was appointed. In 1898 a much better building was erected but it was totally inadequate and not cheerful. The organization was expanded and improved in 1902 when physicians and nurses were appointed who had experience in the state service. Treatment was very greatly extended and advanced.

In 1933 the present fine new building was opened for this service. It has been planned with a view to adequacy for some years as regards size and facilities. When fully equipped it will have over 600 beds. Classification of patients can be carried out satisfactorily so that there will not be the clashes of personality and discrepancies of standards that occur when quarters are inadequate. The largest provision is for the frank psychoses but the service also carries the prison wards and a great many cases that in former days must have been crowded into services that were not able to give the needed type of attention.

The building is nine stories high, providing accommodations for both patients and staff. It is well equipped for the various special therapies and includes medical and surgical units and laboratories. No patient with a mental disorder need be neglected in New York City. In all cases the welfare of the patient is first considered and questions of residence and financial capacity are secondary.

More than 18,000 patients were received in this hospital in the last calendar year and given whatever type of treatment seemed desirable. A very large number of these patients pass on to state hospitals within a few days. Many others go to their homes in New York and elsewhere. Others are sent to whatever type of institution is suitable. There is an out-patient department serving 8000 cases in a year.

Kings County Hospital

Lexington Avenue subway to Winthrop Street. Walk out Clarkson Avenue to Albany Avenue. 9 miles.

Years ago the former City of Brooklyn established an observation service in the Kings County Hospital. After amalgamation of the greater city this service was extended to cover the Borough of Queens also. The psychiatric services at Bellevue and Kings County Hospitals are now operated conjointly. Kings County Hospital received 4480 patients last year. The service occupies two buildings. Many of the patients go from here to state hospitals; those who are infirm need only to be transported across the street to the Brooklyn State Hospital.

New York City Children's Hospital.

Lexington Avenue subway to 125th Street. Surface car eastbound to ferry. 4 miles.

The New York City Children's Hospital is on Randall's Island. This property has been owned by the city since 1835 and has been used for several eleemosynary purposes. All the children for whom the city was caring were moved to it in 1848. Two years later there were reported about 40 mental defectives among the 1396 inmates. As time went on greater provision was made for defectives so that in 1878 they numbered 211 out of 989 children and adult inmates. Since 1915 only mentally defective children have been accepted and the school facilities of the institution have been considerably developed. The admission rate has run from 600 to 1200 a year. The census, which was over 1500 in 1930 is now under 1000 because of construction operations for a new bridge that will lessen the accommodations.

Grasslands Hospital.

New York Central Railroad to White Plains or Valhalla. 23 or 26 miles. Notify Superintendent and car will meet you. Highway: Bronx River Parkway to Valhalla or Saw Mill River Parkway to Eastview. 28 miles. This trip may be combined with visit to Bloomingdale Hospital.

Westchester County, adjacent to New York City on the north, has a population of a half million. The county maintains a large and diversified institution known as Grasslands

Hospital only four miles from the county seat in White Plains. This hospital has for its psychopathic institute a new building opened in 1930. This is a three-story structure of 82 beds, carefully planned for suitable classification of the patients and well equipped not only for care but also for the treatment of its patients. Most of these patients remain but a few days and are then returned to their homes or to the courts or are sent to state hospitals for longer treatment. The service is active, 1118 patients having been admitted in 1933.

Sing Sing Classification Clinic.

New York Central Railroad to Ossining. 30 miles. Highway. Bronx River Parkway to Briarcliff, routes 404 and 133. 35 miles.

In 1917 there was established in Sing Sing Prison a Clinic, the purpose of which was to study the individual prisoner in greater detail than could possibly be done by the prison personnel, to examine all new admissions and to make recommendations that would aid the warden in assigning these men to the proper type of work, in determining their suitability for parole and in helping in every possible way to bring about their rehabilitation. The clinic was at that time supported on private funds and its work terminated during the war. At a later time the state took up the same plan and established the present Classification Clinic in 1927. The three psychiatrists examine all new admissions, over 1700 in a year. They attend the intramural court and the meetings of the assignment board; they dispose of persons who do not properly belong in the prison on account of mental disorder or defect. 1729 new cases and another thousand old cases were seen again in the last year. Considerable psychotherapy is undertaken. The prison personnel are instructed in various phases of their work. The methods of diagnosis and staff conference have thus been adapted to the needs of another type of institution.

Clinic, Court of Domestic Relations.

Lexington Avenue subway or surface car to 23d Street. 1½ miles.

The first court clinic in New York was organized in 1917 for the Children's Court. When this court was merged into the Court of Domestic Relations the clinic followed and its

field of activity was broadened. The primary and principal work of the clinic is still with the children and in the year 1933 cases to the number of 1847 were examined, together with 26 adults. Since 12,000 cases a year pass through the Children's Court it will be seen that 7 per cent of all the children are referred to the clinic for an opinion. The usual variety of problems is found among the cases that the judges refer. The clinic is at 137 East 22d Street near Irving Place. Its work is carried on not only here but also in the children's courts of the other four boroughs of the city.

Psycho-Educational Clinic.

Lexington Avenue subway to 28th Street. 1 mile.

The Board of Education of New York City has for over 30 years maintained ungraded classes for those children who are so far below normal in general intelligence that they cannot profit by a curriculum organized for the average child. These are classes for older girls, older boys and younger children. The class has a work shop and the children beside getting such academic work as they can master are engaged in purposeful activity. The work of the Division of Ungraded Classes under the direction of the late Elizabeth Farrell attained considerable note.

A psychopathic clinic was established on October 20, 1913 in this division, its purpose being to analyze the nervously abnormal children. Later the name of the organization was changed to the Psycho-Educational Clinic. Though located in Manhattan at 224 East 28th Street it also has branches in two other boroughs.

Bureau of Child Guidance.

Lexington Avenue surface car to 57th Street. ½ mile.

Further to develop the understanding and treatment of children who do not fit smoothly into the scheme of school life the Board of Education established in June, 1931, a Bureau of Child Guidance. A year later its clinic division began to function. A district including 28,000 school children was chosen to be fully provided for by this clinic before additional work was undertaken. This district furnishes about two-thirds of

the cases examined; the other third come from the rest of the city. The clinic is organized with psychiatrists, psychologists, social workers, a visiting teacher and consultation facilities, and a full clinical service is given. It functions at 228 East 57th Street.

Clinic of the Court of General Sessions.

Lexington Avenue subway to Worth Street. 4 miles.

In January, 1932, began the operations of the Psychiatric Clinic of the Court of General Sessions. This is located in the Criminal Courts building near the prison called "The Tombs." After conviction and prior to sentence the mental and physical make-up of convicts is studied and reported to the court, to the probation bureau and later to whatever penal institution the prisoner enters. Recommendations for disposition are offered. The clinic is a branch of the psychiatric service in the Department of Hospitals, which operates also the services in Bellevue and Kings County hospitals. In a year 2500 patients have been examined.

Psychoanalytic Institute.

Subway from 50th Street at Broadway, to 86th Street. 2 miles.

The New York Psychoanalytic Institute was made possible by the educational trust fund of the New York Psychoanalytic Society. It began active operation in October, 1931. Its activities are similar to those of the institutes in Berlin, Vienna and London and in order to benefit by the experience of these centres a visiting director was called from Germany. Lectures, seminars and analyses are given here. The institute is housed in a comfortable building at 324 West 86th Street.

Jersey City Hospital.

Newark City Hospital.

Hudson Tube to Jersey City or Newark.

Both these hospitals maintain active psychiatric services for the residents of their neighborhoods. Outpatient clinics also are maintained.

Essex County Hospital.

Subway to Chambers Street. "Tube" to Jersey City, change to train for Cedar Grove. 20 miles. Highway: Holland Tunnel, Route 9 to Verona, 23 to Cedar Grove. 22 miles.

In New Jersey several counties maintain their own institutions for the care of the mentally sick. The largest and best equipped of these is the Essex County Hospital at Cedar Grove. It grew out of an unsatisfactory situation at the State Hospital, which was seriously overcrowded. In 1871 some temporary quarters were erected in the City of Newark. In 1896 the institution was moved to the present 300 acre site where it is housed in a series of buildings, none of too great size, and well differentiated in style and function.

Hudson County Hospital.

Bus from Times Square. 10 miles.

Hudson County also has its own institution. Most of the patients in it are residents of Jersey City. There are two branches; one is on Laurel Hill and the other two miles north at Secaucus.

New Jersey State Hospital at Greystone Park.

Lackawanna Railroad to Morristown or Morris Plains. Taxi. 31 miles. Highway: Holland Tunnel, routes 25 and 24. 31 miles.

Near Morris Plains is the New Jersey State Hospital at Greystone Park, opened in 1876. This institution, like others of that period, received the attention of Dorothea Lynde Dix, who presented 100 books to the patients' library in 1880. The institution has grown in size by the construction of new groups of buildings rather than by extension of the original group and some of the later buildings were especially studied to meet the needs of particular groups of patients. Altogether the patient population has reached 4400.

Veterans Hospital.

Central Railroad of New Jersey. Highway: Holland Tunnel, routes 25 and 24. Trip may be combined with visit to Greystone Park or Skillman.

The newest of the United States Veterans' Administration Facilities is located near Lyons. Many of the patients are from New York City.

Epileptic Village.

West 23d Street ferry. Philadelphia and Reading Railroad. 45 miles. Highway: Holland Tunnel; at Airport right on Route 29 to Somerville, 21 to Belle Mead, local road to Skillman. 50 miles.

The New Jersey State Village for Epileptics at Skillman was opened in 1898. It is situated on a tract of over 1000 acres. Its activities cover all those lines that experience shows likely to promote the well-being of the inmates. The cottage plan has been followed in construction.

New Jersey State Hospital at Trenton.

Pennsylvania railroad. 60 miles. Highway: Holland Tunnel and Lincoln Highway to Trenton. 60 miles.

The New Jersey State Hospital at Trenton is a place of considerable interest in the history of psychiatry. Moved by such sorry conditions as the chaining of insane patients in jails and poor houses, the state established this institution in 1845. There had been the usual long period of discussion but Dorothea Lynde Dix entered the field and threw the legislature into action. It was opened in 1848. Miss Dix was always very proud of this successful campaign and in her later years lived in an apartment there up to her death at the age of 85. On several occasions the hospital has been the beneficiary of legacies from private donors. It was for 26 years the place where the late Doctor Henry A. Cotton carried on the bold studies that have resulted in improved treatment of the physical disorders of the mentally sick throughout this country and elsewhere. The hospital is especially well equipped for surgical activities.

Hartford Retreat.

N. Y., N. H. & Hartford Railroad from Grand Central Station, 98 miles. Highway: Park Avenue and Boston Post Road to New Haven, turn left for Hartford. 110 miles.

The Connecticut State Medical Society had agitated for better care for the mentally sick for 10 years before the charter for the Hartford Retreat was granted in 1822, and in two more years the institution was ready for occupancy. The close economy characteristic of that commonwealth was shown in

the fact " that the whole amount expended on the building, exclusive of the steps and iron sashes, falls a little short of $6622," as reported in January, 1824. Since the buildings were to accommodate 40 patients this makes a per capita construction cost of $314 per patient, a rate that would amaze us today. The reports of the period contain many interesting items, such as that in 1830 two dollars per week was a possible rate if the patient did not remain in the institution over six months. Many additions were made to the original plan, some being extensions of the first building and others being cottages about the grounds. Among the superintendents, Doctors Butler and Stearns were presidents of this Association.

Dorothea Lynde Dix commended the improvements to the institution in 1858 but thought there were fire hazards in the warming and lighting systems. All public patients were removed in 1868. The institution has recently developed what is known as its Psychiatric Institute for acute disorders. There have been many recent changes in organization and the service is extremely active, about twice as many patients being admitted in a year as there are beds. A very active program has been developed embracing physical activities and cultural interests.

Institute of Human Relations.

N. Y., N. H. & Hartford Railroad from Grand Central Station, 72 miles. Highway: Park Avenue and Boston Post Road. 75 miles.

The psychiatric work of the Yale University School of Medicine is centered in the Institute of Human Relations. This is a combined organization of several sciences and attempts to study patients from many different approaches. The building has been in use only about three years. Its psychiatric section represents an effort to provide many desirable facilities for a few patients at a time under study.

Fairfield State Hospital.

N. Y., N. H. & Hartford Railroad to Bridgeport, bus to Newtown. Highway: Bronx River Parkway, Route 22 to Armonk, 128 to Mount Kisco, 117 to Katonah, 22 to Brewster, 6 to Newtown. 75 miles.

The Fairfield State Hospital at Newtown, Connecticut, is a very new institution and at present accommodates only about

500 patients. When completed this attractive and carefully planned hospital will serve all the western end of Connecticut and receive patients on request from other sections.

There follows a list of other organizations that maintain psychiatric clinics under various names. These are listed by the borough in which they are located.

Bronx.

Lebanon Hospital. Westchester and Cauldwell Avenues.

Brooklyn.

Brooklyn Bureau of Charities, 285 Schermerhorn Street.
Brooklyn Hospital, DeKalb Avenue and Ashland Place.
Brooklyn Juvenile Protective Association, 4 Court Square.
Cumberland Hospital, N. Portland Avenue and Auburn Place.
Jewish Hospital of Brooklyn, Prospect Place and Classon Avenue.
Methodist Episcopal Hospital, 6th Street and 7th Avenue.
Polhemus Memorial Clinic, L. I. College Hospital, Amity and Henry Streets.
St. Charles Hospital, 281 Hicks Street.
St. Mary's Hospital, 1298 St. Marks Avenue.
United Israel-Zion Hospital, 4810 Tenth Avenue.
United Jewish Aid Societies, 1095 Myrtle Avenue.

Manhattan.

Bellevue-Yorkville Health Demonstration Center, 325 East 38th Street.
Catholic Charities Guidance Institute, New York Foundling Hospital, 181 East 68th Street.
Clinic for the Social Adjustment of the Gifted, 778 Education Building, New York University, Washington Square, East.
Community Church Memorial Dispensary, 244 East 38th Street.
Community Hospital Dispensary, 8 St. Nicholas Place.

Consultation Service for Students of Teachers College, Dept. of Psychiatric Education, Teachers College, Columbia University.

Consultation Service for Students of Union Theological Seminary, Broadway and 120th Street.

Girl Guidance Bureau, Girls' Service League of America, 138 East 19th Street.

Hastings-Hillside Hospital, Room 504, 67 West 47th Street.

Home Bureau, Hebrew Sheltering Guardian Society, 139 West 126th Street.

Jewish Board of Guardians, 228 East 19th Street.

Jewish Social Service Association, 67 West 47th Street.

Judson Health Center, 237 Thompson Street.

Mount Sinai Hospital, 100th Street and Madison Avenue.

Mount Sinai Hospital, 1 East 100th Street.

Neurological Institute of New York, Fort Washington Avenue and West 168th Street.

Neuropsychiatric Service for Employees of the Metropolitan Life Insurance Company, Medical Division, 1 Madison Avenue.

New York Association for Improving the Condition of the Poor, 105 East 22d Street.

New York Infirmary for Women and Children, 321 East 15th Street.

New York Postgraduate Hospital and Medical School, 303 East 20th Street.

New York Protestant Episcopal City Mission, 38 Bleecker Street.

Pre-School Dept., Greenwich House, 27 Barrow Street.

Seward Park Guidance Bureau, Seward Park High School, Ludlow and Grand Streets.

State Charities Aid Association, 105 East 22d Street.

St. Luke's Hospital, 421 West 114th Street.

Vanderbilt Clinic, Broadway and 168th Street.

Queens.

St. John's Long Island City Hospital, 12th Street and Jackson Avenue.

Richmond.

Staten Island Hospital, Castleton Avenue, Tompkinsville, Staten Island.

There is appended a list of private licensed institutions in the metropolitan district.

For the Care and Treatment of Mental Diseases.

Dr. Bolton's Home, James R. Bolton, M. D., Beacon, N. Y.
Crichton House, Robert B. Lamb, M. D., Harmon-on-Hudson, N. Y.
Dr. Combes' Sanitarium, E. T. Murray, M. D., Corona, L. I.
Craig House, C. J. Slocum, M. D., Beacon, N. Y.
Four Winds, Chas. I. Lambert, M. D., Katonah, N. Y.
Greenmont-on-Hudson, Edmund J. Barnes, M. D., Ossining, N. Y.
Halcyon Rest, Henry W. Lloyd, M. D., Rye, N. Y.
Hastings Hillside Hospital, Louis Wender, M. D., Hastings-on-Hudson, N. Y.
Interpines, Frederick W. Seward, M. D., Goshen, N. Y.
Louden-Knickerbocker Hall, Robert A. Olson, M. D., Amityville, L. I.
Long Island Home, L. G. Price, M. D., Amityville, L. I.
Dr. MacDonald's House, Charles W. Pilgrim, M. D., Central Valley, N. Y.
Pinewood Sanitarium, Joseph Epstein, M. D., R. F. D. Katonah, N. Y.
River Crest, Wm. Elliot Dold, M. D., Astoria, L. I.
Dr. Rogers' Hospital, Henry W. Rogers, M. D., 345 Edgecombe Avenue, N. Y. City.
Stony Lodge, P. R. Vessie, M. D., Croton Dam Road, Ossining, N. Y.
St. Vincent's Retreat, James M. O'Neil, M. D., Harrison, N. Y.
Dr. Wellington's House, W. J. Wellington, M. D., Mamaroneck, N. Y.
West Hill, Henry W. Lloyd, M. D., West 252 Street, New York City.
White Oak Farm, H. E. Schorr, M. D., Pawling, N. Y.

Dr. Barnes' Sanitarium, Frank H. Barnes, M. D., Stamford, Conn.
Blythewood Sanitarium, S. Harcourt Peppard, M. D., Greenwich, Conn.
Stamford Hall, Frank W. Robertson, M. D., Stamford, Conn.
Fair Oaks, Thomas P. Prout, M. D., Summit, N. J.

For the Care and Treatment of Mental Defect.

Bailey Hall, Rudolph S. Fried, Katonah, N. Y.
Brooklyn Home, Sisters of the Congregation of the Daughters of Wisdom, Port Jefferson, L. I.
The Brunswick Home, C. L. Markham, M. D., Amityville, L. I.
Gary de Vabre Academy, J. Gary de Vabre, Lake Ronkonkoma, L. I.
Sound View School, Jennie M. Berault, Yorktown Heights, N. Y.
Sycamore Farm School, Natalie Brewster, Newburgh, N. H.
The Bancroft School, E. A. Farrington, M. D., Haddonfield, N. J.

Notes and Comment.

PSYCHICAL RESEARCH.—A new testing ground for the claims of spiritualism has been organized in London in the form of an international institute for psychical research which proposes to conduct its investigations of alleged spirit manifestations by rigidly scientific procedures and tests. The personnel of the new institute is guarantee of the integrity of its intention. It includes Professor G. Elliot Smith, president, Sir Charles Sherrington, Professor E. D. Adrian, Professor E. W. MacBride, Dr. David F. Fraser-Harris, and Professor Julian Huxley.

We seem to recall that it was Houdini who admonished that the magician rather than the scientist is the expert best qualified to investigate the phenomena of spiritualism.

FOLIA PSYCHIATRICA ET NEUROLOGICA JAPONICA.—The first issue of this new publication appeared in December, 1933, under the editorship of Professor T. Wada, of Osaka, with the collaboration of 16 neurologists and psychiatrists in various centers in Japan. On the editorial staff Professor Uyematsu, of Tokyo, has supervision of clinical neurology, Professor Saito, of Tokyo, of clinical psychiatry, Professor Nakamura, of Niigata, of psychology, and Professor Hayashi, of Okayama, of anatomy, histopathology and biochemistry.

The Folia will appear quarterly approximating 600 pages per annum. Contributions will be published in German, English or French. The present number contains eight articles in German, and one in English.

The contents of Volume 1, No. 1, are:

1. Ohyama, K.: The Effect of Malaria Treatment on Pupillary Disturbances in Progressive Paralysis.
2. Kubo, K., and Hattori, R.: Progressive Paralysis in Korea.
3. Uyematsu, S., Fujii, Y., and Kamano, H.: Fever Therapy of Progressive Paralysis by means of Intravenous Injection of Gono-Vaccin.
4. Kubo, K., and Hikaru, N.: Statistical Studies on 120 cases of Facial Palsy.

5. Takeuti, M., and Namba, Y.: Pathological Changes in the Central Nervous System in Lead Poisoning.
6. Takeuti, M.: Experimental Investigation of Fibre Connections between the Thalamus and the Area Retrosplenialis, particularly the Thalamocortical Fibres.
7. Satô, S.: Experimental Investigation of the Localization of Visual and Auditory Functions in the Cortex of Guinea Pigs and Cats.
8. Kamimura, T.: Distribution of Neuroglia in Human and Animal Spinal Cords.
9. Takeuti, M.: Experimental Investigations of the Fibre Connections between the Thalamus and the Area Retrosplenialis. II communication.

The JOURNAL extends greetings to the editors of the Folia Psychiatrica et Neurologica Japonica and wishes them every success.

MENTAL HOSPITAL STATISTICS IN CANADA.—There has recently appeared the first annual report for the calendar year 1932, compiled by the Dominion Bureau of Statistics covering the activities of all mental institutions in Canada. This report, which is issued in both English and French, makes readily available a great variety of statistical information concerning the 58 institutions existing in Canada January 1, 1932, which include 28 public hospitals for mental disease, mental defect and epilepsy; 3 public hospitals for the mentally deficient; 3 training schools for feebleminded children; 2 psychiatric hospitals; 16 county and municipal institutions; 4 private sanitaria; and 2 psychopathic hospitals administered by the Department of Pensions and National Health.

The report shows that there were on the books of 56 institutions on December 31, 1932, 35,279 patients, of whom 19,498 were males, and 15,781 females, including 1989 patients on parole. The total increase in the number of resident patients during the year 1932 was 1249.

The total admissions during the year were 9790 (first admissions 7307, readmissions 1886, transfers 597). The sex ratio for first admissions was 135.3 males to 100 females; for readmissions 113.3 males to 100 females.

The ratio of discharges per 100 admissions for all Canada was 50.5. By discharge is meant that the patient is written off the books of the hospital and released from supervision by the hospital authorities. Of these 50.5 discharges per 100 admissions for 1932, 14.2 were reported recovered, 22.2 improved, 10.6 unimproved.

The total number of discharges during 1932, exclusive of transfers and deaths, was 4948. Of these 1391 (28.1%) were discharged as recovered, 2175 (43.9%) improved, 1034 (20.8%) unimproved, 330 without psychosis, and 18 unclassified. The deaths occurring in hospitals totalled 2325.

WORK OF THE COMMONWEALTH FUND.—The fifteenth annual report for the year ending September 30, 1933, indicates that the Commonwealth Fund expended for the public welfare for the 12 months' period $1,662,714. About a fifth of this expenditure was devoted to unemployment relief in New York; nearly a half of the total went into public health, rural hospital services, and medical education and research. One of the outstanding publications was "A Standard Classified Nomenclature of Disease" prepared by a conference of representatives of the principal national medical associations, and which was referred to in a previous number of the JOURNAL. Among the research projects assisted by the Fund during the year were studies in rheumatic fever, dental caries, trachoma, multiple sclerosis, kidney structure and function, and the normal development of children.

The Commonwealth Fund has been particularly interested in the field of psychiatry and mental hygiene, and in its report calls attention to the action of The American Psychiatric Association in establishing a certifying board to examine physicians who wish to qualify in psychiatry, and also to the two year study of psychiatric education conducted by the National Committee for Mental Hygiene. The report continues:

> As psychiatric education is taken more seriously in medical schools there may be a growing demand for qualified teachers. It is partly with this need in view that the fund has provided fellowships for graduate psychiatric study at Phipps Psychiatric Clinic, Boston Psychopathic Hospital, and the Colorado Psychopathic Hospital, and has given two new fellowships this year at Pennsylvania Hospital, Department for Mental and Nervous Diseases, in cooperation with the Philadelphia Child Guidance Clinic.

In addition scholarships have been continued for training in psychiatric social work at the New York School of Social Work, at Smith College, at Western Reserve University, and at the University of Chicago.

During the period of operation of the New York Institute for Child Guidance, which was closed during the year, the report shows that formal training was given to 32 psychiatrists, 15 psychologists, and 289 psychiatric social workers, and clinical services to 2641 children.

In London a Child Guidance Clinic, supported chiefly by the Fund, has completed its fourth year. Its value has been recognized by the London County Council, which, under the provisions of a new law, proposes to utilize its services for the examination of children who are held in custody awaiting court action.

Association and Hospital Notes and News.

REPORT OF NOMINATING COMMITTEE.—In accordance with the provisions of the revised constitution the following nominations are announced to be acted on at the annual meeting of The American Psychiatric Association, to be held in New York City, May 29–June 1, 1934.

For President: Dr. C. F. Williams, Columbia, S. C.
For President-elect: Dr. Clarence O. Cheney, New York, N. Y.
For Secretary-Treasurer: Dr. Wm. C. Sandy, Harrisburg, Pa.
For Councillors for 3 years: Dr. Geo. H. Kirby, New York, N. Y.; Dr. Forrest C. Tyson, Augusta, Me.; Dr. Glenn Myers, Los Angeles, Cal.; Dr. Winfred Overholser, Boston, Mass.
For Auditor: Dr. Albert C. Buckley, Philadelphia, Pa.

(Signed) JAMES V. MAY, *Chairman,*
EARL D. BOND,
ADOLF MEYER,
SAMUEL T. ORTON,
WILLIAM L. RUSSELL.
Nominating Committee.

AMERICAN PSYCHOPATHOLOGICAL ASSOCIATION MEETING.—The twenty-fourth annual meeting of the American Psychopathological Association will be held in Atlantic City, Sunday night, June 3, and Monday, June 4. The American Neurological Association also will hold its annual meeting in Atlantic City, beginning Monday, June 4. Members of the Neurological Association are cordially invited to the meetings of the Psychopathological Association.

The exact time and place of meeting will be announced later. Titles of papers should be in the hands of the Secretary, Dr. L. E. Emerson, not later than May 1.

Secretary's address: 370 Commonwealth Ave., Boston, Mass.

PROGRAM 1934 MEETING.—Dr. Hamilton, Chairman of the Committee on Program, announces that the program for the forthcom-

ing annual meeting will include a joint session with the Association for the Study of Mental Defect and a joint session with the Section on Psychoanalysis. Several symposia are being arranged. The topics include: "The significance of some Common Hospital Functions"; "Psychiatry in Medicine and Surgery"; "Endocrine and Vegetative Nervous System Aspects of Psychiatry." A number of studies in Pathology have been offered and various miscellaneous papers.

The Sections on Convulsive Disorders and on Forensic Psychiatry and Conduct Disorders will each hold two sessions on Monday preceding the general meeting. The Section on Psychoanalysis will hold a meeting on Wednesday afternoon.

The following matters will probably be discussed by round table groups:

College Mental Hygiene.
Criminology.
Functional Psychoses in Children.
Occupational Therapy.
Psychiatric Nursing.
Psychiatric Social Work.
Psychoanalytic Therapy.
The Treatment of Excitement.
Veterans Administration Problems.

CONVENTION TRAVEL THROUGH CANADA.—We have been advised by the National Parks of Canada of the Department of the Interior at Ottawa, that prospective delegates to our forthcoming annual meeting and their friends, who wish to travel through Canada en route to the convention city or to visit that country following the meeting, can obtain from that office information which will be of assistance to them in planning such a trip, including maps showing the main automobile roads between the United States and Canada, booklets on various phases of recreation in Canada, and a helpful folder entitled "How To Enter Canada." Considerable unpublished data is also available to those making known their specific requirements. Enquiries may be directed to J. B. Harkin, Esq., Commissioner, National Parks of Canada, Dept. of the Interior, Ottawa, Canada.

MEETING OF AMERICAN ASSOCIATION ON MENTAL DEFICIENCY.—The annual meeting of the American Association on Mental Deficiency will be held at the Hotel Waldorf-Astoria, New York, May 26, 27, 28 and 29, 1934. The Saturday session, May 26, will be given over to the sociological, psychological and the special educational aspects of the problem in order that local social workers and school teachers may have an opportunity to attend without interfering with their regular duties. The Tuesday afternoon session will be a conjoint meeting with the American Psychiatric Association. Data as to the program may be obtained from the Secretary, Dr. Groves B. Smith, Godfrey, Illinois.

THE SURGEON GENERAL'S CONFERENCE ON PUBLIC HEALTH.—The Surgeon General of the Public Health Service held an informal conference at his office on January 30, 1934, for the purpose of discussing the immediate and future needs of public health services in the United States. Members of the National Advisory Council of the Public Health Service, representatives of official and non-official organizations interested in public health, and members of the Surgeon General's staff attended the conference.

Mental Health was represented not only by officials of the United States Public Health Service but by Dr. Clarence M. Hincks, Director, National Committee for Mental Hygiene, and Dr. Arthur Ruggles, President of the American Foundation for Mental Hygiene, and Chairman, Scientific Administration Committee of the National Committee for Mental Hygiene.

The conference began at 10 a. m. and lasted throughout the day, the discussion embracing both international and national aspects of public health. The national aspects had special reference to the integration of federal, state and local health agencies; educational requirements and policies; and research.

PORTRAIT OF DR. SALMON FOR NEW YORK PSYCHIATRIC INSTITUTE.—On January 26, 1934, the Thomas William Salmon Memorial Committee presented a bas-relief portrait of the late Dr. Salmon to the New York Psychiatric Institute and Hospital, Columbia-Presbyterian Medical Center. The portrait was accepted on behalf of the State of New York by Dr. Frederick W. Parsons, Commissioner of the Department of Mental Hygiene.

The placing of this memorial in the Psychiatric Institute commemorates the services of Dr. Salmon to his city and state in the fields of mental hygiene and public welfare. It was he who first proposed that New York State, Columbia University and the Presbyterian Hospital should cooperate in establishing a State Psychiatric Institute and Hospital as part of the Columbia-Presbyterian Medical Center. Dr. Salmon was the first director of the National Committee for Mental Hygiene, and was at the time of his death in 1927 professor of psychiatry at Columbia University.

The memorial tablet, executed by Charles Keck, New York City, presents a portrait of Dr. Salmon in profile, and includes the following inscription:

Professor of Psychiatry, Columbia University, 1921-1927. Beloved Physician, Teacher, Mental Hygiene Leader, Whose Vision Guided the State and the University in Placing Here This Psychiatric Institute and Hospital.

Book Reviews.

THE MENTAL DEFECTIVE. By *Richard J. Berry, M. D.* and *R. G. Gordon, M. D.* (New York: Whittlesey House, McGraw-Hill Book Company, 1931.)

In view of the large number of individuals who are called upon to deal with mental defectives and who have no special training in the problems of mental deficiency, the authors set out to "epitomize something of the real achievements made in the recent study of mental deficiency and the underlying development of the growing brain." They do so in the belief that "social inefficiency" is the greatest problem of the modern state.

Mental deficiency is defined as "a partial or complete arrest of brain growth and development, especially of the minute cells of the brain, which occurs either before birth or at some time between birth and the age of puberty. In consequence of this arrest, the individual is unable to react to his environment in the manner regarded as normal by the average member of society." This arrested development may be inherent in the germ plasm or may occur during the early developmental stages. Mental defectives show many departures from the normal stages of development.

Legal recognition of mental deficiency began in England with the Act of 1899 and has gradually progressed as far as the Act of 1927. It is pointed out that the various levels of mental deficiency are not clearly demarcated one from the other, as the law suggests, but rather shade into one another. Similarly there is no rigid line of demarcation between the defective and the normal. The actual standard for normalcy varies with the varying complexity of the social environment. The real problem, the authors point out, is social inefficiency. Social inefficiency may be due to defective will control or abnormal instincts without mental defect. That this problem of social inefficiency is one of great import is indicated by the fact that in the United States 1100 million dollars is expended upon law and protection as against 600 million expended upon schools and churches.

The authors would appear ready to have us forget what they have already stated, that social inefficiency is not synonymous with mental defect. Still less can the total cost of the administration of the law be laid at the door of the defective.

The lack of an absolute standard for diagnosing mental defect has been a large factor in causing the estimates of the incidence of mental deficiency to vary, as they do, from 4.61 per thousand to 8.00 per thousand. The authors, themselves, suggest that with rigid standards the incidence might not improbably be .15 or .20 per cent. What these rigid standards are is not stated.

The methods at present used—segregation and sterilization—have not successfully coped with the problems, the authors state. We shall search in vain, however, for any further solution offered us in this book.

In a chapter on the evolution of the brain, the human brain is compared with that of various animal forms. The mental defective brain is deficient in the number of brain cells, particularly in the distinctively human supra-granula layer. The infra-granular layer, which is connected with animal and instinctive reflexes, is actually enlarged in the mental defective, accounting for his greater tendency to anti-social behavior. Microcephaly, which is generally a sign of a deficient number of brain cells, occurs in 75 per cent of all cases of mental defect. Microcephaly is here apparently used to denote head size at or below the 10th percentile of the general population. The deficient number of brain cells tends to cause disconnection in neuronic arcs, which, the authors believe, predisposes the individual to aberrant forms of behavior.

Mind is defined as "the sum total of all our mental processes, which are themselves only different functions of the nervous system, especially the brain." Intelligence is hard to define but however defined it is something in which the mental defective is lacking. It is a sufficient number of brain cells correctly linked. A brief behavioristic account is given in which simple reflexes, instincts, emotions, habits, and voluntary actions are all described in terms of stimulus and response.

In many ways the defective varies from the normal. Head size tends to be diminished. It is, however, the number of neurons, not brain size and weight, which correlates with intelligence. Defectives tend to have diminished physique and reduced vitality. There is delay in the acquisition of normal functions, such as, control of bladder and rectum, walking, talking. Defects in sight, hearing, and dentition are often associated with mental deficiency. Epilepsy, and abnormal nervous manifestations occur frequently. The instincts are not controlled by integration of the cognitive and emotional phases, hence defectives are unmoral rather than immoral. From their ranks are recruited many criminal, prostitutes, and social inefficients. This then is "the greatest of human degeneracies, the most menacing of national evils."

Defectives are classified legally as idiots, imbeciles, and feeble-minded. The idiot is speechless and helpless. The imbecile is described as able to do simple jobs under supervision but "unable to do sufficient work to contribute to his own support." This seems to indicate a difference in standards from those used on this continent. Certainly many whom we consider in the upper strata of the imbecile group are able to contribute very materially to their own support, under supervision. The feeble-minded are socially inefficient in a complex society. Medicine divides defectives into clinical groups such as the mongolian, cretinoid, syphilitic, etc. These, the authors believe, are not very significant as the causes in most instances are not really known and treatment is obscure and ineffective.

There are two groups of factors in the etiology of mental deficiency—heredity and developmental factors. Primary amentia is due to causes

operating on the fertilized ovum, most probably due to defective germ plasm. This group apparently comprises the majority of defectives. Secondary amentia is due to causes operating at or after birth, such as birth injury, fevers, etc. An exposition of the Mendelian laws of heredity follows together with the statement that the evidence indicates that 80 per cent of mental defect is hereditary in origin. This latter is a bold statement in view of the fact that most recent studies have thrown grave doubt on any such high estimate of the part played by heredity.

Aments fall into two groups, from another point of view, social and asocial (or as they are called in the United States, stable and unstable). The social are the most numerous. The asocial show mal-praxis chiefly in the two major groups of instinct, the self-preservative and reproductive instincts. It is this which makes the asocial ament a national problem. Such mal-praxis occurs, however, amongst those who are not mentally deficient.

Mental defect is related to other forms of illness. The defective's low vital capacity predisposes him to illness. The poor calibre of his neurons makes him tend to suffer from organic nervous disease and in addition handicaps him in being retrained to compensate for lost nervous tissue. In the causation of mental illness there are three groups of factors—brain structure, temperament, and emotional conflict. In brain structure the defective is inferior; in temperament he shows similar variations to those of the normal; as far as emotional conflict is concerned he is comparatively free as his ideals are not sufficiently organized to cause a great deal of conflict. Thus from some aspects the defective has greater tendency to mental illness and from some aspects less. The hysterical form of reaction is more common in defectives, whilst anxiety neuroses are less so. "True" epilepsy the authors would like to regard as a special form of mental defect rather than as something associated with the defect. Mental defect, because of the lack of vitality of neurons, is closely related to dementia.

We should not be unduly alarmed by the rapid post-war increase in the incidence of mental deficiency, as such a condition existed after the Napoleonic wars and is an inevitable consequence of the depletion of the best breeding stocks. A more permanent tendency for increase in mental defect is brought about by the curbing of nature's law of the survival of the fit through social legislation. This is coupled with an apparently deliberate restriction of size of family among the upper strata of society. In view of the increasing complexity of social life any increase in the proportion of low mentalities seems highly undesirable. On the other hand, machine production is doing away with the necessity of skill thus making a place for the high grade defective in industry. The social consequences of mental deficiency are, however, very grave. The authors state that there is "a large amount of scientific evidence to prove the association of feeble-mindedness with crime, poverty, and prostitution. The defective is responsible for the slums. Alcoholism and drug addiction are more frequent among defectives. Truants and vagrants are usually defective."

In discussing a policy for the problem the authors bewail the excessive expenditure on the unfit. In a program for dealing with the defective early

diagnosis is essential. This diagnosis should not be merely on an educational basis. The ability to "fend for oneself" should be the crucial test. Segregation is the next step. The model colony is a little village inexpensive in construction and as nearly as possible self-supporting. It should be provided with a medical and psychological staff. Borderline cases should be left under "deferred diagnosis" until such time as it can be definitely decided as to whether segregation is necessary. Sterilization has been effective as a partial solution.

In pursuing their objective of educating the general public with regard to the problem of mental deficiency the authors have supplied much useful information. In some respects, however, their position does not appear to be clearly worked out. They have rightly recognized that "social inefficiency" is the important problem. They have also stated that social inefficiency may occur without mental defect and that mental defect may occur without social inefficiency. They have not been clear in this, however, for at times they speak as though the two problems were identical. In this respect the book is confusing. While pressing for careful and scientific consideration of these problems they make many statements such as that the slums are due to defectives, alcoholism and drug addiction are more common among defectives—statements for which they give us no statistical authority at all.

Whether or not the reader is willing to accept the physiological psychology of this book, he can scarcely rest content with it as an explanation of the problems of the book. That mental deficiency is due to deficiency in the number of brain cells is seriously questioned to-day. The explanation of asocial behavior is even less acceptable. An interesting characteristic of the defective is the enlargement of the infra-granular cortical layers (connected with instinctive and animal desires) together with diminished supra-granular layers (connected with reasoning and control). But this does not account for the fact that some defectives are asocial while others are not, or for the fact that asociality occurs in non-defectives. In their attempt to give an understanding of mental deficiency and social inefficiency the authors have been too ready to put forward over-simplified explanations of what neither they nor anyone else have as yet been able to explain adequately.

The book throws into relief many aspects of the problem of mental deficiency. It performs a timely service in pointing out the inadequate provision for the problem. It cannot be said however that the presentation is always clear or that the perspective is always true.

S. J. W. Horne.

General Principles of Human Reflexology. An Introduction to the Objective Study of Personality. By *V. M. Bechterev.* English Translation by *Emma* and *William Murphy,* from the Russian fourth (1928) edition. (New York: International Publishers, 1933.)

For at least 30 years Russian students of mental function have insisted, more consistently than those of other countries, on a strictly objective viewpoint for psychology. They have shown a preference for physiologic

methods in the investigation of mental phenomena and, in extreme cases, have objected to the use of such words as memory, association, judgment, and the like, maintaining that until these phenomena can be understood and expressed in physiologic terms they cannot be counted as scientific data but merely as problems demanding investigation.

This conception of the tasks of psychology has resulted, on the one hand, in a series of epoch-making researches of the higher nervous activity of animals, and, on the other hand, in a significant meagerness of contribution to our knowledge of human mental function.

Among the leaders in this field must surely be counted the author of this book, the late Professor Bechterev.

One of the foundation stones of Bechterev's teaching is a recognition of two kinds of reflexes: simple and association-reflexes. The latter kind is apparently identical with Pavlov's conditioned reflexes and would seem to be the elements of what psychologists would ordinarily regard as mental function.

Curiously enough the term conditioned reflexes is scarcely referred to in the book in spite of the obvious fact that the researches on conditioned reflexes carried on by the Pavlov school are practically the only foundation for "a science of reflexology," if there be such a thing.

Pavlov and Bechterev, the two principal Russian leaders in the investigation of cerebral function, are remarkably contrasted personalities. They are representatives, respectively, of the two types of great men in science described by Ostwald (in his book "*Grosse Männer*"), *Klassiker* and *Romantiker*.

Bechterev's activity has been characterized by an amazing multiplicity of ideas and active participation, both under the old régime and since the revolution, in the establishment of various institutions, the founding of organizations, societies, journals, and a million irons in the fire in general.

In the biographical sketch written for the book by Professor Gerver, a disciple of Bechterev, we find: "During the last 30 to 40 years there has been no congress of psycho-neurology, pedology, or pedagogics in which Bechterev has not directed the proceedings and delivered addresses of general interest." Also, "Practically no students' meeting of importance was held in the Psycho-neurological Institute at which Bechterev, whom the students always received with marked enthusiasm, was not present." And, further, "As Bechterev considered hypnosis particularly useful in the treatment of alcoholism, he introduced a collective method of hypnotizing patients and secured splendid results."

Pavlov has been content to work out with the most painstaking care details of cerebral function with the aid of experimentally developed conditioned reflexes of salivary secretion in the dog and to point out casually a possible bearing that such researches may have eventually on problems of human cerebral function and on practical problems in psychiatry. On the other hand, Bechterev, with a much more meager basis of research, could not resist the temptation of venturing upon free speculation and proceeding

naively not only to the establishment of a new science, "human reflexology," but also of a second new science, "collective reflexology," to take the place of what is more commonly spoken of as sociology—though without such claims of a scientific position for it.

Nothing is farther from the reviewer's mind than any thought of belittling the achievements of the Russian workers in the field of physiologic psychology. And it is not necessary to put any limit whatever on our hopes or dreams of further achievements that may be made in this field in the future. But it would seem that only a complete detachment from the realities of the situation and an abandonment to wish-fulfilling fancies could have led a person to imagine, as actually accomplished, such vast applications of reflexology to pathology of personality, psychiatric therapeutics, the elucidation of phobias, sexual perversions, vocational aptitudes, the field of pedagogy, "reflexology of the masses," etc., as are mentioned by Bechterev.

Perhaps a couple of quotations from the concluding chapter in the book will give the reader some idea of the general impression that it produces throughout:

"In the investigation of personality, not only must attention be paid to medical examination and to investigation of the general constitution, to the anthropological type, to various bio-chemical and, in particular, hormonic and fermentative reactions, to ordinary or inherited reflexes, but we must also take into consideration all the manifestations of personality, under the various forms of association reflexes and dominant processes or concentration, in their relationship with the external and internal stimuli, both present and past. For this purpose, we have elaborated a scheme to guide observation of the person's main reactions—under the particular environmental conditions and in response to the various environmental influences—in respect of his manifestations in the sphere of complex-organic reflexes or instincts, in the mimico-somatic sphere, in the sphere of established behavior sets in general, in respect of the character of his speech reactions and of his peculiar creative activity. Uninterrupted observation of the person's manifestations and their causes or stimuli, even over a period of 10 days, is sufficient to enable us to arrive at a more or less complete determination of his relation to the environment and of his reactions dependent on various influences. Missing links may be filled in by natural experiment, including the artificial application of an appropriate stimulus, and we utilize this experiment in such cases. Here, of course, an analysis of the reaction is necessary; not, however, the subjective analysis in vogue among psychologists, but a correlation of the person's various manifestations one with the the other; a correlation of aggressive, inhibitory, and defensive reactions to certain stimuli, these reactions being results of past experience and established set; a correlation with the age, sex, constitution, anthropological type, special talent, etc. Observation of the professional work also and of the results achieved in it is of the utmost importance. This necessitates special methods of observation and investigation. Every such investigation demands, of course, some expenditure of time, but always provides valuable material for the study of personality."

"Next we should determine the person's capacity for work, that is, his exercisability, the development of excitation during the given work, subsequent fatigue, as well as the degree of inner effort and the stability of concentration on the work (by means of applying an irrelevant stimulus at a certain moment). Lastly, speed in the formation and differentiation of association-motor reflexes must be investigated by laboratory methods. In addition, the individual peculiarities of creative activity in given conditions of work may be investigated by special methods.

"All these indications may be utilized only in reference to the objective data, and we must not exclude creative activity, which is to be evaluated in respect of time, form, expression of personality, greater or lesser success in the solution of the task, the character of the work, the relation to the environment, etc.

"It is always essential to consider not only the quantitative, but also the qualitative, aspect of the work, and to do so perfectly objectively, without excursions into the subjective sphere. Thus, in their connection with hereditary conditions, with peculiarities of the physical constitution and general reactivity connected with hormonic conditions discoverable by medical, anthropological, and bio-chemical investigations, the essential aspects of the personality of man as an agent in the social environment may be revealed, when his behavior has been studied over a certain period of time."

A. J. R.

EXPERIMENTS WITH HANDWRITING. By *Robert Saudek*. (New York: William Morrow & Co., 1929.)

This is a most exhaustive study of the principles, methods and doctrines of experimental graphology. Since Abbe Michon 70 years ago began to critically examine writing with the idea of setting out certain signs which appeared to reflect psychical characteristics, there have emerged many metaphysical graphologists whose vague phrases have served to divest their works of scientific respectability. The author throws himself into this breach and sets out to prove that his contribution offers a basis for a new and exact science and for basic reforms in forensic graphology. He consigns to microphotography, palimpsest photography and chemical investigations, physical alterations and physical changes in writing, such as erasures and superimpositions. Having thus allotted his field he discusses most thoroughly the following:

I. The Development of the Graphic Faculty from Earliest Childhood to Complete Graphic Maturity.
II. The Relative Speed of the Act of Writing.
III. Attention and Spurious Expression in Handwriting.
IV. The Central Nervous System and the Act of Writing.
V. Individual Features of the Handwriting and their Symptomatic Significance.

VI. The Graphic Signs of Honesty and Dishonesty.
VII. The Method to be Followed in the Characterological Analysis of a Handwriting.

Throughout, the author in a psychological approach, emphasizes the postulate that writing is essentially a brain process, reflecting the mood, character and visual memory of the writer—stressing this rather than the mechanical act. The fact that a visual memory is the most potential auxiliary in the imitation of written forms is shown by the result of experiment in which children write more rapidly typographic forms than cursive script. The psychology of the fear complex in writing is of especial significance to the psychiatrist. Fluent and automatic writing may be slowed up, inhibited or distorted in the latter, the word or the complete sentence. Many complexes arise because as the author observes "we may speak as it is natural for us to speak, but we must write only as a pedantic schoolmaster would write who wanted to display his acquired knowledge in carefully turned sentences from which all colloquial accents have been excluded." This comparative formalism in writing must be carefully considered in any interpretative study. A must illuminating glossary, explanatory notes and an illustrated supplement of handwriting is appended.

JOHN EISELE DAVIS.

MENTAL HYGIENE IN THE COMMUNITY. By *Clara Bassett.* (New York: The Macmillan Company, 1934.)

"Mental Hygiene in the Community," published in January, 1934, is written by one trained and experienced in psychiatric social work. It is a panoramic exposition of available data aiming to present in brief and correlated form the various ways in which mental hygiene may be of importance and value, and where it may profitably function in the present organization of community life. The book should stimulate interest and encourage experimentation for meeting local mental health needs and situations. Each chapter contains pertinent suggestions for preliminary study and investigation of specific phases of the problem and illustrations of methods that are applicable for experimentation.

The author views the present status of mental hygiene as being unable to offer definite and concrete solutions for the many problems involving the amelioration, treatment, or cure of the multiple adverse mental and personality situations or their prevention; or the promotion of positive mental health, the latter depending on forces lying partly outside the question of illness and its avoidance. The contributions that law, psychology, sociology, education in its broadest sense, or public policies must make in the latter field cannot avoid the fundamental place which modern medicine occupies or may be expected to occupy in the future, for the promotion of good mental health implies a knowledge of factors influencing the nature, causation, and prevention of ill health. Her approach to these yet unsolved problems augers a healthy appreciation of the present status of mental hygiene when her facile

pen depicts the situation thus: "From the standpoint of pure science, mental hygiene is hardly out of its swaddling clothes and many more decades of research and experimentation will be necessary before such a happy consummation will be achieved. The evolution of every science and art has been a slow fumbling and stumbling toward the light of truth; and mental hygiene is no exception to this rule. The only development possible is through the conscientious application of the best knowledge available at the moment, followed by a keenly critical appraisal of the accumulated experience and results obtained, which in turn brings about modifications in theory and practice so that these are ever more closely in accord with the facts and needs of the situation. Through this process mental hygiene will gradually emerge from the haze of conflict which envelops the various schools of thought, its theories will be more precisely defined, clarified, consolidated and tested and a more and more effective instrument for the study, treatment and prevention of mental and behavior disorders will be forged."

The book, comprising twelve chapters with index and list of bibliographies, lends itself to convenient assignments in reading courses or text study. The first chapter involves an introduction to the subject; the second a discussion of medicine with special reference to the recognition that every form of illness has a psychic component, and to the need for graduate and undergraduate instruction. The third chapter discusses the position of nursing in the field; the fourth the place of social service agencies, followed by other chapters devoted to the subjects of delinquency and law, parental education, the pre-school child, education and teacher training, the church and theological training, industry, recreation, and psychiatric institutions and agencies.

W. L. T.

AN OUTLINE FOR THE PSYCHIATRIC CLASSIFICATION OF PROBLEM CHILDREN. By *Sanger Brown, II, M. D.*, Assistant Commissioner of Mental Hygiene, *Horatio M. Pollock, Ph. D.*, Director of Mental Health Statistics, and *Howard W. Potter, M. D.*, Assistant Director, Psychiatric Institute and Hospital. (Utica, New York: State Hospitals Press, 1933.)

This 16-page booklet is worthy of serious consideration by all physicians connected with children's psychiatric clinics for it attempts "to provide for the orderly grouping of the various types of mental disorders in children." It points out that "the mental deviations presented by problem children are not as fixed or as clearly defined as the mental disorders seen in adult patients. The terminology used in diagnosing adults, therefore, can be used only to a limited extent in classifying children." All types of children seen in various clinics may be classified within this outline and a very skilful grouping exists for those "in which environmental situations constitute the only problem." The notes on diagnostic procedure, the general instructions and the definitions and explanatory notes are especially valuable. It is interesting to compare the threefold diagnostic grouping of psychiatric classification, physical classification and psychological classification with the system in use in the Ontario

Mental Health Clinics. We offer the suggestion that as most physicians are used to "physical diagnosis," that this classification might come first. Eight main groups of disorders are given in this outline as contrasted with 10 groups suggested by Dr. Gillespie in his article on "Psychology and Psychopathology of Childhood" in the British Medical Journal of November 15, 1930.

E. P. Lewis,
Toronto.

Major Aspects of Personality. By *Maurice H. Krout.* (Chicago: The College Press, 1933.)

This book is the product of a misapprehension. The author has, in his own words, "tried to bring together the contributions of biology, anthropology, sociology and psychology and weld them together to form a theory of personality." But it is not so that theories are formed, and one is not surprised that no new theory emerges. The various sciences which treat of personality do so each with its own concepts and frame of reference in mind, and hence the forms which the data ultimately assume are inherently disparate and not reducible to each other. Prof. Krout has failed to achieve any synthesis, not because he lacks adequate acquaintance with the terminology and assumptions of the various disciplines, but because he appears to have thoughts of their findings as "contributions" which have only to be assembled to give a "true" picture of personality. Whereas the different approaches are to a great extent mutually exclusive, and reconcilable only in terms of some higher synthesis which we shall need a Darwin to suggest.

The misapprehension is most obvious in the final chapter, which is a general outline for the study of personality in the form of a gigantic schedule or inventory. This schedule, exclusive of a large "Summary and Diagnosis" section, contains no less than 784 headings, all of which must presumably be taken into account in any given case. While some of these items require merely a tick to denote presence or absence, others refer to scores on lengthy personality tests, many will require at least a paragraph of carefully analyzed observations, (A. VII. A. 4. b. "responses to parental successes, failures, illnesses"), and some particularly in the ecological, biological and cultural sections, represent extensive projects of study in themselves. (*e. g.,* A. III. C. 4. d. "individual's attitude towards community as observed in conversation, tension states, and overt behavior.") Such a scheme, regarded as a systematization of a vast number of concepts used in the analysis of personality, is both interesting and instructive; but, as the author admits, it is frankly unusable clinically. And a point which appears to have escaped his attention is that the same item of behavior may, owing to the variety of the approaches, appear in several sections under entirely different names.

Prof. Krout's understanding of science is largely in terms of objectivity, and much space is given to the discussion of the conditioned reflex as the fundamental mechanism of personality formation. All modification is referred

to conditioning processes, and as these are often highly obscure, the term is used with the looseness too familiar in behaviorist writings. And the barbarous terminology which appears whenever the "psychic" is treated objectively is conspicuously present; "love-object," for example, is "positive co-conditioned stimulus." All pretence of objectivity, however, evaporates when the author reaches the section on personality development, and the Freudian dogmas of identification, ambivalence, castration-fear and the like are paraded in as bald and challenging a way as the present reviewer has ever met with. Prof. Krout claims a detached attitude towards psychoanalysis; but if that be the case it is incomprehensible that pages 228 to 239 should ever have been printed in their present form.

There is, however, a good deal of interesting material in the book, particularly the author's own work on symbolic gestures, which is well presented and illustrated, and is to form the substance of a later work. The criticism of the trait-psychologists, and the discussion of unconscious and subconscious behavior in terms of the lack of verbal equivalents, are also valuable.

The book is very poorly printed, and studded with extraordinary misprints, while the chapter headings in the body and in the appendix do not correspond. And the author himself is often careless in his writing; it is very doubtful, for example, that Thorndike (or anyone else!) ever asserted that man has a tendency to pursue small animals and to "tear them *from limb to limb*" (p. 92).

J. D. KETCHUM.

BEHIND THE DOOR OF DELUSION. By *"Inmate—Ward 8"* (New York: Macmillan Co., 1933).

This book will prove of interest to those unfamiliar with mental hospital conditions, and should be of real value in dispelling many false or exaggerated beliefs concerning the work of these institutions. The picture given by the author is undoubtedly a fair one, and represents the situation in many state hospitals.

The author is a newspaper man, and in this book he does not depart from the journalese style. While, for reasons of his own, he has avoided sensational statements at the expense of the institution in which he was confined, one cannot but wonder whether the undercurrent of paranoia, which his writing contains, is not merely another way of appealing to the morbidness of his readers. His discussions of the woman "Constance," whom he finally decides to marry, are irrelevant and in poor taste, whether they be inserted to supply "human interest" or are bye-products of egoism.

The author does not pretend to have more than observational acquaintance with psychiatry, and his occasional digressions into this field are naturally amateurish. To the lay reader, however, they might give the impression of a competence which he does not possess.

FRANCIS FIELDING-REID,
Baltimore, Md.

L'INTUITION DÉLIRANTE. By *R. Targowla* and *J. Dublineau*, under the direction of *Drs. Henri Claude* and *J. Levy-Valensi*. (Paris: Editions Médicals Norbert Malvoine, 1931.)

The purpose of the authors in presenting this study is to describe for the psychiatrist a hitherto undefined symptom—*l'intuition délirante*, found in many mentally ill persons. An attempt is made by the writers to ignore the usual psychological and metaphysical connotations of the term "intuition." A three-fold plan of elucidation is adopted, (1) positive definition or statement concerning the nature of the symptom; (2) manifestation of the symptom and its relationships in more than 60 detailed case studies; (3) negative definition or differentiation of the symptom from certain clinical entities with which it is often closely associated.

Positively, *l'intuition délirante* is an immediate judgment, not requiring proof, and which appears to exhibit absolute certainty to the patient. Negatively, it is distinguished from hallucinations and psychic-hallucinations as these have "exterior" relationship or significance; it is differentiated from psychotic interpretations which are not direct judgments; it fails to have the inventiveness of psychotic imaginations. Often "l'intuition" is found as a basis for certain of these entities or closely associated with them. The symptom is found in patients having anxiety states, also in hypomanics, schizophrenics, and psychasthenics.

The case studies are well selected for the purpose intended—to reveal the importance of the described symptom in psychic disturbances. They illustrate to a certain extent the rôle played by *l'intuition délirante* in the reactions of the patients. The complexity of the thought processes in such persons is evident.

The work, which is largely speculative, is the result of extended studies. It is of value in attempting a better understanding of such phenomena as psychic hallucinations and fixed ideas. The study marks a forward step in exploring the thinking of mentally ill persons.

WILFRED SCOTT.

THE LIBRARY OF HISTORY OF DIODORUS OF SICILY, VOL. I. Translated by *C. H. Oldfather*, Professor of Ancient History and Languages, University of Nebraska (New York: The Loeb Classical Library. G. P. Putnam's Sons; London: William Heinemann, Ltd., 1933.)

If it is good diet for the soul to turn away sometimes from "the discouraging life of a volcanic present," as Feuchtersleben hath it, "toward the quiet regions of the inner man," so may it be after another fashion, in the pages of history to recapture the meaning of things past. Among the December issues of the Loeb Classical Library is a book to the purpose, volume I of the Universal History of Diodorus Siculus.

It was the purpose of Diodorus to recount the annals of all the known world from mythologic beginnings down to his own day, in the consulship of

Julius Cæsar. Thirty years, he tells us, he devoted to the task, during which he visited the principal countries of the Mediterranean world about which he was writing. It is his theme that the study of history should be the means to the good life and to this end he stresses in the lives of his characters the consequences good and bad of their courses of action. We glimpse that relentless and inevitable figure in Greek tragedy, Fate, who infallibly punishes and rewards the deeds of men, soon or after many years.

"It is fitting," writes Diodorus, "that all men should ever accord great gratitude to those writers who have composed universal histories, since they have aspired to help by their individual labours, human society as a whole; for by offering a schooling which entails no danger, in what is advantageous they provide their readers, through such a presentation of events, with a most excellent kind of experience. For although the learning which is acquired by experience in each separate case, with all the attendant foils and dangers, does indeed enable a man to discern in each instance where utility lies, yet the understanding of the failures and successes of other men, which is acquired by the study of history, affords a schooling that is free from actual experience of ills. Furthermore, it has been the aspiration of these writers to marshall all men, who, although united one to another by their kinship, are yet separated by space and time, into one and the same orderly body. And such historians have therein shown themselves to be, as it were, ministers of Divine Providence."

The history of Diodorus included 40 books, of which 15 are extant. The present volume contains Book I, which deals with the myths, kings and customs of Egypt, and a portion of Book H, which treats of Assyria, India, Scythia, Arabia Felix, and the islands of the ocean.

His story of Egypt is a fascinating chronicle compiled from his own observations and enquiries in that country and from the narratives of earlier historians. As the translator of the present work remarks, it is "the fullest literary account of the history and customs of that country after Herodotus." He was perhaps better qualified than Herodotus to gain reliable information because at the time of his visit, Greek, the language of Diodorus, had been the official tongue of Egypt for nearly 300 years.

In the story of Diodorus, compound of fact and fable, we learn of the beginnings of the universe and of gods and of men. In the beginning the sun's rays directly generated in the soft primeval earth every form of animal life. Only later when the earth had become solid and therefore "no longer able to generate any of the larger animals," was it necessary for the perpetuation of living creatures that they should breed one with another.

"The first men to be born, they say, led an undisciplined and bestial life . . . . the sounds which they made were at first unintelligible and indistinct, yet gradually they came to give articulation to their speech. . . . . But since groups of this kind arose over every part of the inhabited world, not all men had the same language . . . . and out of these first groups to be formed came all the original nations of the world.

"Now the first men, since none of the things useful for life had yet been discovered, led a wretched existence. . . . . Little by little, however, experience taught them both to take to the caves in winter and to store such fruits as could be preserved. . . . . Indeed, speaking generally, in all things it was necessity itself that became man's teacher.

"Egypt is the country where mythology places the origin of the gods, where the earliest observations of the stars are said to have been made. . . . . Men first came into existence in Egypt, both because of the favourable climate of the land and because of the nature of the Nile. . . . . As proof that animal life appeared first of all in their land they would offer the fact that even at the present day the soil of the Thebaid at certain times generates mice in such numbers and of such size as to astonish all who have witnessed the phenomenon."

We are told of the origin of the first gods, Osiris the many-eyed sun, and Isis the moon whose birth was from everlasting and who wore horns to symbolise both the crescent moon and the cow sacred to her.

Diodorus sets forth the circumstances, as they leaked out from the priests, of the mysterious death of Osiris, and of the secret disposal of his body by Isis; of the origin of phallic worship and the mysteries of Dionysus; of the law permitting brother-sister marriages, and of the custom whereby husbands at marriage agreed to obey in all things their wives; of the exemption of the priests from taxation, and of the origin in Egypt of the ritual of circumcision; of the introduction of castes. He explains the curious means contrived by nature for the benefit of man to keep down the numbers of crocodiles.

We are told how Rameses II gained immortality in stone, his monolithic statue, "largest of any in Egypt" weighing 1000 tons; and of his challenge to posterity: "King of Kings am I. If any one would know how great I am and where I lie, let him surpass one of my works." The Ramesseum at Thebes is described in detail. In a bas-relief of a court scene the judges are shown "without any hands, and in their midst the chief justice, with a figure of Truth hanging from his neck and holding his eyes closed and at his side a great number of books. And these figures show by their attitude that the judges shall receive no gift and that the chief justice shall have his eyes upon the truth alone."

We are made acquainted with the legendary king Sesostris who after a beneficent reign of 33 years committed suicide, with the approval of both priests and populace, in that "he had caused the end of his life to comport with the loftiness of spirit shown in his achievements."

Diodorus records a peculiar test by which the chastity of the women of Egypt was tried; he reports the building of a labyrinth which, it is said, was later copied by Minos in Crete; he describes the conjectured means by which the pyramids were raised.

But we cannot extend indefinitely the references to this most readable history. We close with the account of the manner of life of an Egyptian king. "All their acts were regulated by prescriptions set forth in laws,

not only their administrative acts, but also those that had to do with the way in which they spent their time from day to day, and with the food which they ate. . . . . And the hours of both the day and night were laid out according to a plan, and at the specified hours, it was absolutely required of the king that he should do what the laws stipulated and not what he thought best. In the morning he had first of all to receive the letters which had been sent from all sides. . . . . Then after he had bathed and bedecked his body with rich garments and the insignia of office, he had to sacrifice to the gods. . . . . When the king had performed the divination from the entrails of a calf and had found the omens good, the sacred scribe read from the sacred books the counsels and deeds of distinguished men, in order that he who held the supreme leadership should first contemplate the most excellent general principles and then turn to the prescribed administration of the several functions. For there was a set time not only for holding audiences or rendering judgments, but even for his taking a walk, bathing, and sleeping with his wife, and, in a word, for every act of his life. It was the custom for the king to partake of delicate food . . . . and drinking only a prescribed amount of wine, which was not enough to make them unreasonably surfeited or drunken. Their whole diet was ordered with such continence that it had the appearance of having been drawn up, not by a lawgiver, but by the most skilled of their physicians, with only their health in view.

C. B. F.

Die Psychopathischen Persönlichkeiten. By *Prof. Dr. Kurt Schneider.* Third revised edition. (Leipzig und Wien: Franz Deuticke, 1934.)

The first edition of this work (a brochure of 96 pages), was issued in 1923 as one of the sections of Aschaffenburg's Handbuch der Psychiatrie, the first encyclopedia of psychiatry, with which Bumke's Handbuch der Geisteskrankheiten now disputes the field.

During the five years which elapsed between the first and second editions of Schneider's book occurred such an expansion of the field of characterology and personality study, that an adequate survey of this field, still possible in 1923, was no longer feasible in 1928. The auther limited himself more strictly to the discussion of psychopathic personality types from the clinical standpoint.

The same plan is followed in the present edition which has been enlarged to 123 pages. There is a new discussion of the physical basis of psychopathic personality. Personality, normal, abnormal or psychopathic is the necessary and equivalent expression of the indivisible psychophysical constitution of the individual. Schneider adopts Kahn's definition of "constitution": "the totality of the morphological, functional and developmental characteristics in so far as they are transmitted or transmissible by heredity, *i. e.*, inherent in the genotypic structure." The constitution theory of E. R. and W. Jaensch and the localization theories of Kleist and others are incorporated, with increasing stress upon the physical aspect of constitution. The personality concept becomes inevitably more materialistic. For Schneider psychopathic

personality is rigidly a congenital anomaly while he makes allowance for the possibility of psychopathic states due to intrauterine disease or birth trauma, he adds that the term "congenital" applied to abnormal variations of the personality means "almost the same" as inherent in the *anlage*.

Noteworthy is the author's discussion of the relations between psychopathic personality and psychosis, with particular reference to schizophrenia, cyclothymia and epilepsy. He defines schizophrenia as a congeries of psychotic processes which destroy or transform the personality, which are fundamentally irreparable although in widely varying degree, and whose etiology and somatic basis are unknown. Raising the question of gradual transitions as between normal (and psychopathic) personalities and schizophrenia on the basis of analogies from the field of somatic disease, such as are furnished by the gradations of Basedow's disease, the relation of abnormal sugar metabolism to outspoken diabetes, and the transition from essential hypertonus to arteriosclerosis, Schneider concludes that there is insufficient evidence clinically of transition states between abnormal personality and schizophrenia. He is equally positive with regard to cyclothymia, which he defines, in contrast to schizophrenia, as a fundamentally recoverable psychosis comprising depressive, manic or alternating phases and with unknown physical basis. Admitting that theoretically transitions should be possible, he feels that clinical evidence of such transitions is lacking. He speaks nevertheless of mild cyclothymic phases which seem to resemble very closely the clinical pictures of certain psychopathic personalities, and further of periodic or episodic variations which psychopathic personalities may exhibit; these two facts adding to the difficulties of differential diagnosis. However the author has faith in clinical barriers and is disposed to uphold the integrity of discreet clinical pictures.

The classification of psychopathic personalities remains the same in this edition as in the first. The description of clinical types has been revised and augmented as the author's views have developed, and the bibliography has been brought up to date. Schneider's 10 varieties of psychopathic personality are: (1) Hyperthymic psychopaths; (2) Depressive psychopaths; (3) Self-insecure psychopaths; (4) Fanatics; (5) Attention-seeking psychopaths; (6) Temperamentally unstable psychopaths; (7) Explosive psychopaths; (8) Insensitive or antisocial types; (9) Weak-willed psychopaths; (10) Asthenic psychopaths.

C. B. F.

In Memoriam.

L. PIERCE CLARK.

On the afternoon of December 4, 1933, Dr. Clark excused himself from a patient and went to his room to lie down as he was not feeling well. He had some pain in his chest. Two hours later he was found to have died, presumably from a coronary thrombosis.

In this manner the life work of one of the most alert and active members of our special psychiatric fraternity came to a quiet and peaceful close. He would not have wished a more tranquil ending, save for the fact that there was much more to be accomplished and he was but 63 years of age and in the full swing of his productive activity.

Dr. Leon Dennis Pierce Clark was a country boy, born in Ingleside, Steuben Co., New York, on June 15, 1870. Just across the border in Ontario County was the old established Naples Academy. Here Dr. Clark obtained his academic education and then coming to New York he obtained his medical degree at the University Medical School when he was just about 22 years of age.

Following his graduation he served on the City Hospital staff on Blackwells Island, and his very first contribution to medical literature, on May 15, 1894, was a paper read before the New York Academy of Medicine on an epidemic of 40 cases of typhus fever which occurred on Blackwells Island in the first years of his service there.

He then became assistant director and neuropathologist at the Connecticut State Hospital in Middletown. Here we have from these contacts a Study on Aphasia (1894); Observations on Bevan Lewis' method of Preparing Brain Tissue (1894); Tabes Dorsalis in a Woman of 23 (1894); Epicerebral Hemorrhages in General Paresis (1894). From this position he advanced by 1896 to become first assistant physician at the newly founded Craig Colony for Epileptics at Sonyea, New York.

Even before this while at Middletown, Dr. Clark contributed a study on Flechsig's methods of treating psychotic epileptics (1895), which pointed the way to his future interest in this group of phenomena.

It was, however, at Craig Colony that he began the intensive work with the convulsive disorders that has left his stamp on this field ever since. His numerous contributions to a long and valuable series of studies in the epilepsies was begun here and carried out with numerous associates here and later in New York—Sharpe, Tyson, Atwood, Scripture, *et al.* Then he resigned from his work at Craig Colony, went to Europe for a year with Dr. T. Prout, working chiefly with Marburg in Obersteiner's Laboratory in Vienna and with Hughlings Jackson at the Queens Square Hospital in London. He spent a short time in Baginsky's Pediatric Clinic in Berlin. In October 1902 he opened his office in New York City. On the basis of work done on status epilepticus, particularly with Prout, the Stevens Triennel Prize was awarded to them in 1903.

Dr. Clark became a member of The American Psychiatric Association and the American Neurological Association in 1906. In 1907 he read his first paper there, with Atwood on Changes in Type of General Paralysis. Up to this time he had already contributed 22 papers on topics of nervous and mental disease.

In 1908 he contributed his first paper to Psychoanalysis, since which time he devoted much of his interest to psychoanalytic work particularly in the understanding and psychotherapy of the convulsive and compulsive phenomena.

Dr. Clark took active part in various special societies devoted to neuropsychiatry. He was president of the New York Neurological Society in 1911-1912, of the New York Psychiatric Society in 1916, of the National Association for the Study of Epilepsy in 1919 and of the American Psychopathological Association in 1923.

He revised Herter's small work on Diagnosis of Nervous Disorders in 1904, collaborated with Diefendorf on Manual of Methods of Neurological and Psychiatric Diagnosis in 1907. In 1926 he started a quarterly, the Archives of Psychoanalysis, but abandoned it after four numbers were issued.

His volume of poems, entitled "Warp and Woof" shows his deep poetic feeling and his later works on biography his life-long interest in human personality studies, particularly his work on

Lincoln, recently published (1933). For many years, Dr. Clark had directed work in a small institution where he could carry out his psychiatric, psychoanalytic and psychotherapeutic conceptions, particularly with the epileptic personality.

In 1900 while at Craig Colony, he married Elizabeth Holt, and had four children. She died after a severe lingering malignant hepatic illness. He remarried and lost a second wife who died of a cocaine idiosyncrasy while undergoing a minor operation. He also lost an only son who at the age of 24 was drowned. He married a third time and is survived by his widow and two married daughters.

Nowhere in the medical literature is there to be found more intense application to the study of the epilepsies than can be found in Dr. Clark's work. For many years he had been preparing a comprehensive manual on this general group. It is to be hoped that this work of a lifetime may have been left in form for final publication, for in spite of much diffuseness his researches on the epilepsies will constitute a lasting memorial of his work.

Dr. Clark was a stimulating conversationalist. Ideas flowed from his ready mind in rich profusion. He was particularly genial with young people. He was a loyal friend, worked unremittingly in spite of many severe blows dealt him by life's mischances, but he never swerved from his interest in humanity in his medical work.

SMITH ELY JELLIFFE.

DR. WILLIAM H. HANCKER.

Dr. William H. Hancker, superintendent emeritus of Delaware State Hospital died at 9.30 a. m. Friday, December 29, 1933.

Dr. Hancker was a native of Philadelphia and was educated at Girard College, graduating in 1866. He was an apprentice in pharmacy at the Northern Dispensary of Philadelphia under Dr. D. D. Richardson, and graduated from the Philadelphia College of Pharmacy in 1870. He graduated from the Jefferson Medical College in 1873, and became the assistant of Dr. Richardson in the insane department of the Philadelphia Hospital and remained there until 1875.

He was appointed assistant superintendent of the Northern Hospital for the Insane at Winnebago, Wis., in 1875, and remained there until 1882, when he resigned and entered upon the practice of medicine. He was recalled to the Winnebago Hospital and served as assistant superintendent until 1890, when he accepted the appointment of assistant superintendent of the State Hospital at Farnhurst, which post he held for two years. Immediately after the resignation of Dr. D. D. Richardson, his stepfather, Dr. Hancker was appointed as superintendent of the Delaware State Hospital.

On April 1, 1926, Dr. Hancker resigned as superintendent because of ill health. Since then he has served as superintendent emeritus on the staff of this hospital.

Serving as superintendent of Delaware State Hospital for 33 years and being a citizen of Delaware for 43 years, Dr. Hancker was regarded as a great asset to the state as well as to the medical sciences, particularly of that branch of medicine known as psychiatry. He has taken an active interest in all that pertained to the care and treatment of the mentally sick in this state.

Dr. Hancker was a member of many fraternities, including The American Psychiatric Association (life member), American Medical Association, Medical Society of Delaware and the New Castle County Medical Society.

The funeral services were held at the home of Dr. M. A. Tarumianz, superintendent of Delaware State Hospital, on Tuesday January 2, at 11 a. m. Interment was in Philadelphia, Pa.

M. A. TARUMIANZ.